The Journal of Bone and Joint Surgery

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The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association
American Society for Surgery of the Hand
New Zealand Orthopaedic Association
South African Orthopaedic Association
Western Orthopedic Association

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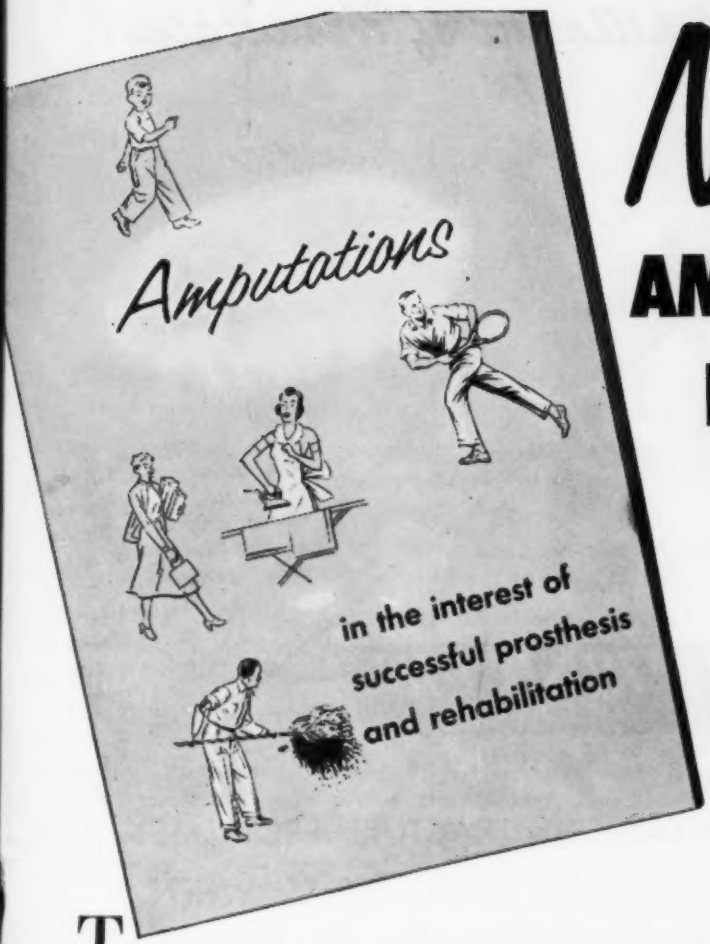

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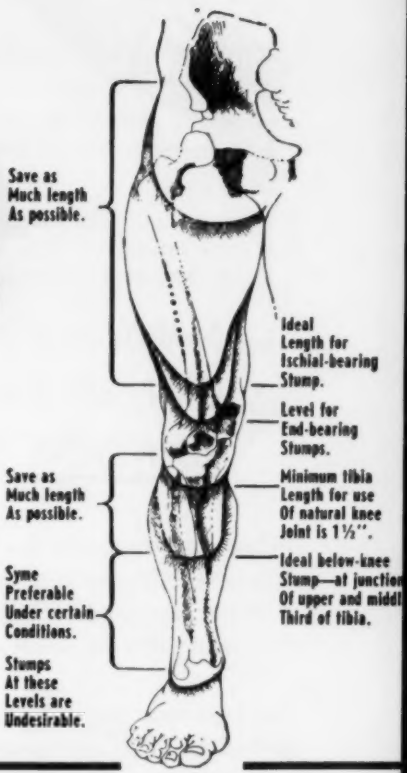

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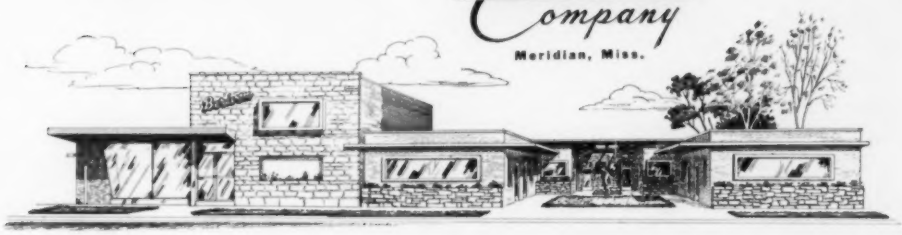

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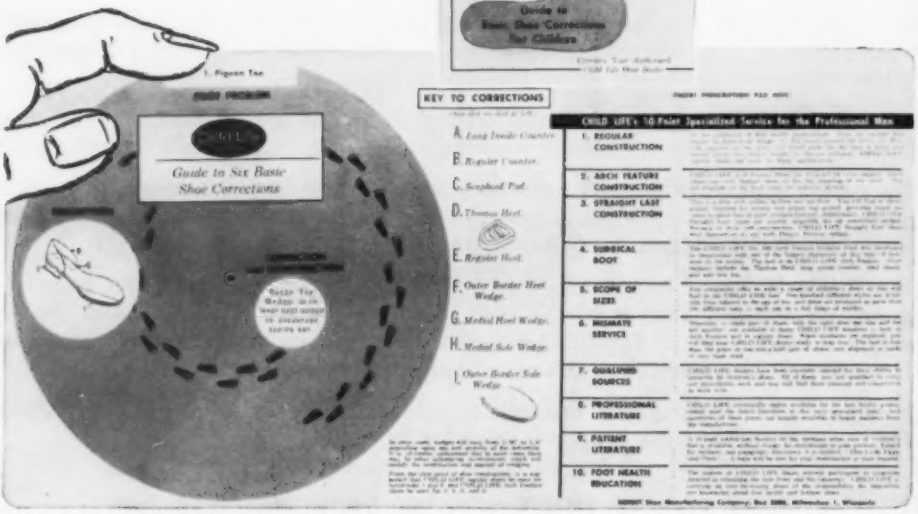

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Degree of Relief from Pain

Degree of Relief	% of Total Patients
Complete Relief	29.4
Most Pain Relieved	49.8
Most Pain Remained	11.5
No Relief	9.3
	100.0

Relief from Pain Compared to Analgesic Previously Used

(Was reported in 1,927, or 68.4% of patients.)

Relief from Pain	% of Total Patients
Superior	60.6
Equal	28.4
Inferior	11.0
	100.0

Degree of Relief from Muscle Spasm

(Was reported in 2,074, or 73.6% of patients.)

Degree of Relief	% of Total Patients
Complete Relief	32.3
Most Spasm Relieved	48.0
Most Spasm Remained	12.3
No Relief	7.4
	100.0

Relief from Spasm as Compared to Previous Muscle Relaxant

(Was reported in 998, or 35.4% of patients.)

Relief from Spasm	% of Total Patients
Superior	53.2
Equal	38.1
Inferior	8.7
	100.0

Undesirable Effects

2,590 patients, or 92.0%, reported no side effects.
226 patients, or 8.0%, reported side effects.

	No. of Patients
Drowsiness	72
Nausea and/or Vomiting	57
Other Gastrointestinal Complaints (Upset Stomach, Heartburn, etc.)	29
Itching, Skin Rash, etc.	15
Vertigo	14
Constipation	6
Miscellaneous	33
	226

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Action

Ethoheptazine (Zactane®) was first synthesized in the Wyeth Institute for Medical Research. The general pharmacological properties and analgesic potency of ethoheptazine in animals have been reported. The clinical effectiveness and safety of this compound have been reported by Grossman, Golbey, Cass, and Batterman and their colleagues. In summary, ethoheptazine is an active analgesic which does not cause sedation, disorientation, constipation, suppression of cough reflex or change in pupil size, nor does it have addiction liability. The combination of ethoheptazine and acetylsalicylic acid (Zactirin®) in the recommended dosage has been found to be equivalent to codeine and acetylsalicylic acid in analgesic potency. The effectiveness and safety of Zactirin for relief of the common types of pain have been reported by several investigators.

The pharmacological action of meprobamate has been carefully studied and described in detail by Berger. This compound, although resembling mephenesin in structure, differs pharmacologically in that its anticonvulsant properties are more profound, its muscle-relaxant effect is of longer duration, it possesses an unusual type of sedative effect not common to either mephenesin or the barbiturates, and, finally, it is active when administered by the oral route. Meprobamate has been proved clinically effective as a skeletal muscle relaxant in patients with neurological conditions, rheumatic disorders, trauma, and as an adjunct to physiatric management of musculoskeletal and neuromuscular disorders. Meprobamate has been shown to be of considerable value in the management of anxiety and tension occurring alone or as an accompanying symptom complex to medical and surgical disorders and procedures.

A combination of meprobamate and acetylsalicylic acid was found by Mitchell to be a useful and safe medication for the treatment of skeletal muscle spasm of varied causes, such as myalgias due to infectious diseases; states of anxiety, tension, and nervousness; premenstrual tension and primary dysmenorrhea; and functional headache. Splitter used EQUAGESIC in patients suffering from musculoskeletal pain associated with muscle spasm and anxiety or tension and found it to be an effective therapeutic agent.

The practicing physician's use of Zactirin administered simultaneously with meprobamate for the relief of muscle spasm and anxiety associated with common painful conditions caused us to prepare the combination oral dosage form. Investigators using meprobamate and ethoheptazine with acetylsalicylic acid tablets have reported this combination to be an effective anti-anxiety, muscle-relaxant analgesic with low side effect liability.

Indications

EQUAGESIC is an effective and well tolerated anti-anxiety, skeletal-muscle-relaxant analgesic and may be used for relief of pain which is accompanied by either skeletal muscle spasm or tension and anxiety or both.

Administration and Dosage

The usual dosage of EQUAGESIC is one or two tablets three or four times daily as needed for the relief of pain and accompanying skeletal muscle spasm or anxiety.

Side Effects

Serious side effects have not been observed following the administration of EQUAGESIC. A small percentage of patients may experience nausea with or without vomiting and epigastric distress. Dizziness occurs but rarely when EQUAGESIC is administered in the recommended dosage. The meprobamate may cause drowsiness but, as a rule, this disappears as the therapy is continued. Should drowsiness persist, this symptom can usually be controlled by decreasing the dose. It may be desirable in some instances to administer central stimulants such as amphetamine or mephentermine sulfate concomitantly to control drowsiness.

On rare occasions, meprobamate has caused severe allergic reactions. This has occurred in most instances in patients who have had only 1-4 doses of meprobamate and have not had a previous contact with the drug. Previous history of allergy does not appear to be related to the incidence of reactions. Mild reactions are characterized by an itchy urticarial or erythematous, maculopapular rash which may be generalized or confined to the groins. Acute non-thrombocytopenic purpura with cutaneous petechiae, ecchymoses, peripheral edema and fever have also been reported. More severe cases, observed only very rarely, may also have fever, fainting spells, angioneurotic edema and bronchial spasm. Treatment consists of the administration of epinephrine, an antihistaminic and, possibly, hydrocortisone or similar agents. Meprobamate should be stopped and reinstitution of therapy should not be attempted.

EQUAGESIC or any of the ingredients used separately in the recommended dosage (Zactane®, Zactirin®, Equanil®) has not been reported to have caused constipation, change in pupil size, disorientation, any significant degree of tolerance, or untoward effects on the formed elements of the blood or cardiovascular system.

Caution

Preparations containing acetylsalicylic acid should be kept out of the reach of children.

Careful supervision of dose and amounts prescribed for patients is advised, especially with those patients with a known propensity for taking excessive quantities of drugs. Excessive and prolonged use of meprobamate in susceptible persons, for example, alcoholics, former addicts, and other severe psychoneurotics, has been reported to result in dependence on the drug. Where excessive dosage has continued for weeks or months, dosage should be reduced gradually rather than abruptly stopped since withdrawal of a "crutch" may precipitate withdrawal reactions of greater proportions than the original indication for prescription. Abrupt discontinuance of doses in excess of the recommended dose has resulted in some cases in the occurrence of epileptiform seizures.

EQUAGESIC should not be given to individuals with a history of sensitivity or severe intolerance to acetylsalicylic acid or meprobamate.

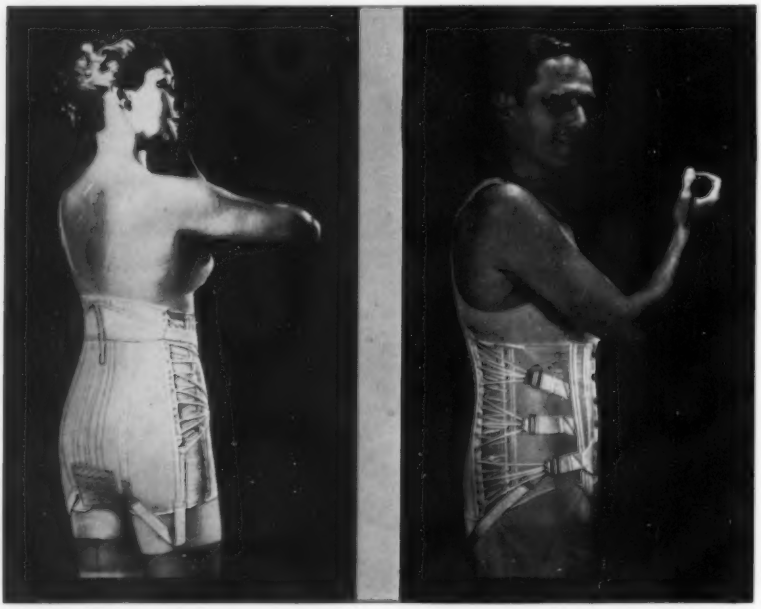
Instances of accidental or intentional significant overdosage with ethoheptazine combined with acetylsalicylic acid (Zactirin) have been reported to produce mild depression, drowsiness, and a feeling of light-headedness, with uneventful recovery. Appropriate therapy of the signs and symptoms as they appear is the only recommendation possible at this time. Overdosage with ethoheptazine combined with acetylsalicylic acid (Zactirin) would probably produce the usual symptoms and signs of salicylate intoxication. Observation and treatment should include induced vomiting or gastric lavage, specific parenteral electrolyte therapy for keto-acidosis and dehydration, watching for evidence of hemorrhagic manifestations due to hypoprothrombinemia which, if it occurs, usually requires whole blood transfusions.

Suicidal attempts with meprobamate have resulted in coma, shock, vasomotor and respiratory collapse. Although up to this time very few of these have been successful in spite of ingestion of large amounts of the drug, doses greater than those recommended may be hazardous. Meprobamate should be given cautiously, and in small amounts, to patients who have suicidal tendencies. In cases where excessive doses have been taken, therapy is symptomatic and may include central stimulants, pressor amines and careful observation. Sleep ensues rapidly after excessive dosage but is normal in character, with blood pressure, pulse and respiratory rates reduced to basal levels.

References available on request

Wyeth Laboratories Philadelphia 1, Pa.

CAMP LUMBOSACRAL SUPPORTS



Camp fabric lumbosacral supports play an important part in the conservative treatment of orthopedic conditions. They steady and limit the motions of the joint, ligaments and muscles in injuries and diseased conditions of the low back. Available without or with steel upright reinforcements or with the Camp spinal brace as needed.

Camp's Lumbosacral Supports are scientifically designed to give a secure fit to the pelvic girdle, the upper lumbar and the low dorsal spine, including the entire abdomen in front, thus giving maximum lumbar spine support with greatest patient comfort.
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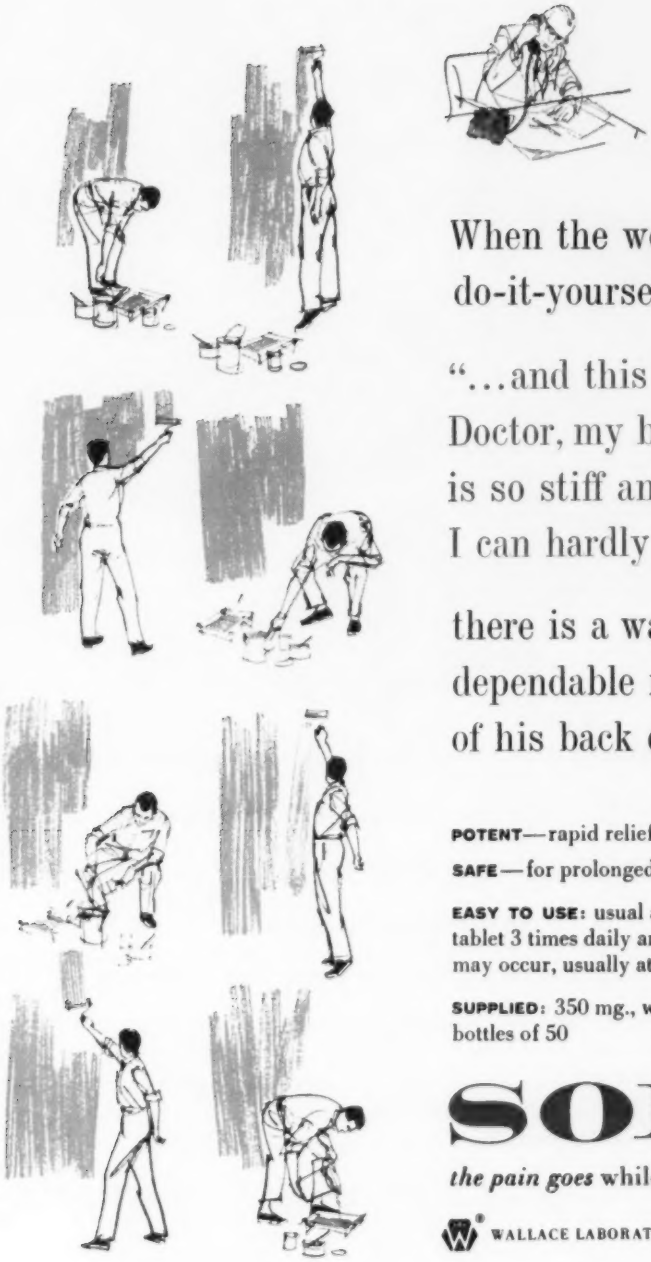

When the weekend
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"...and this morning,
Doctor, my back
is so stiff and sore
I can hardly move."

there is a way to early,
dependable relief
of his back distress

POTENT—rapid relief in acute conditions

SAFE—for prolonged use in chronic conditions

EASY TO USE: usual adult dosage is one 350 mg. tablet 3 times daily and at bedtime (drowsiness may occur, usually at higher dosage)

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the pain goes **while** ***the muscle relaxes***

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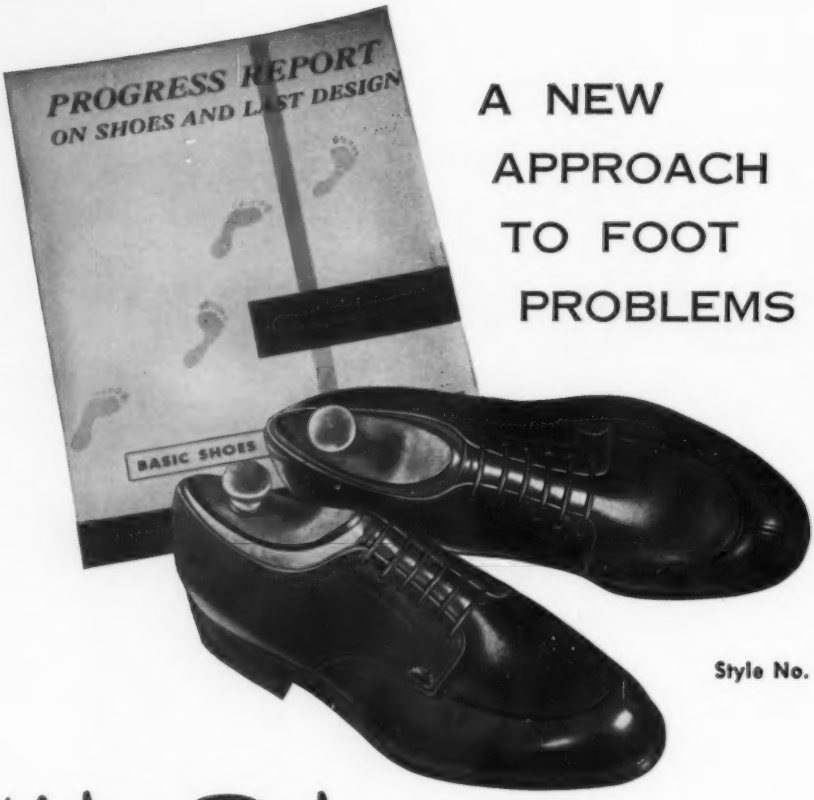

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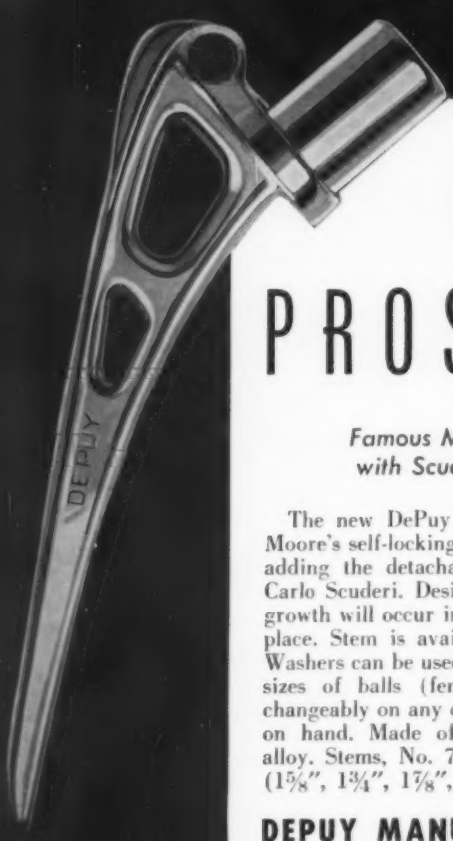

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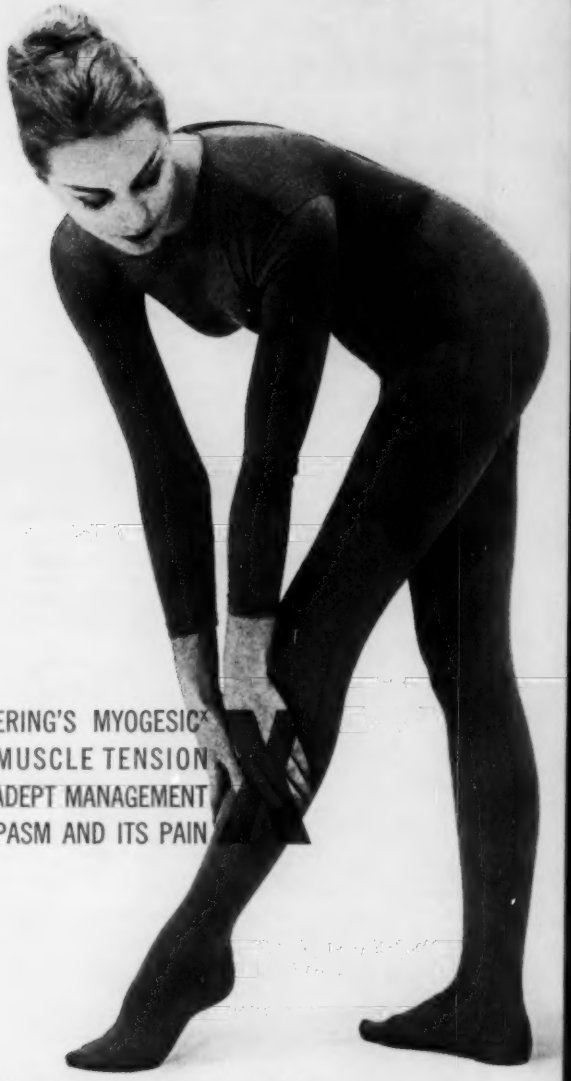

NO SPRAIN, NO STRAIN, OR LOW BACK PAIN

can resist the rapid relaxant relief of

RELA™

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Rela is most useful in the areas where narcotic analgesics are unwarranted and where salicylates are inadequate. Its muscle-relaxant properties are dependable yet significantly free of the limitations or problems often associated with other relaxants.

Rela relaxes acute muscle spasm. Relief of muscle spasm (excellent to good effectiveness in the majority of patients).[1]

Rela provides persistent pain relief through its relaxant and analgesic actions. "Relief from pain was usually rapid and sometimes dramatic."[1]

Rela provides comfort free of spasm and pain. "A number of patients reported freedom from insomnia which they attributed to freedom from pain."[1]

Schering

[X]**MYOGESIC:** MUSCLE RELAXANT ANALGESIC

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1. Kuge, T.: To be published.

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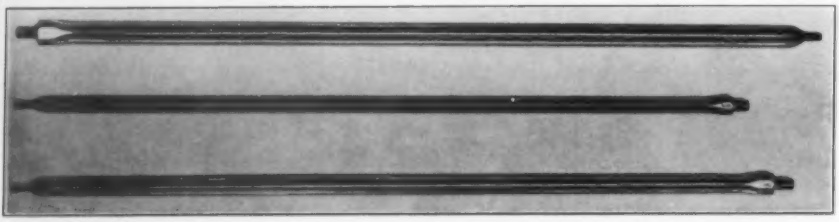
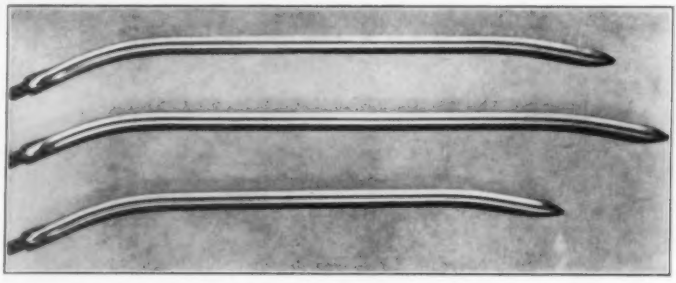


***LOTTES* FEMORAL NAILS** are manufactured with diameters of ⅜, 7/16, and ½ inches —with lengths in ½ inch variations from 14 to 20 inches.

***LOTTES* TIBIAL NAILS** are made in 7/16 and ⅜ inch diameters—in lengths from 9 to 15½ inches, in variation of ½ inch.



***LOTTES* NAILS AND INSTRUMENTS ARE MADE OF 18-8 SMo STAINLESS STEEL**

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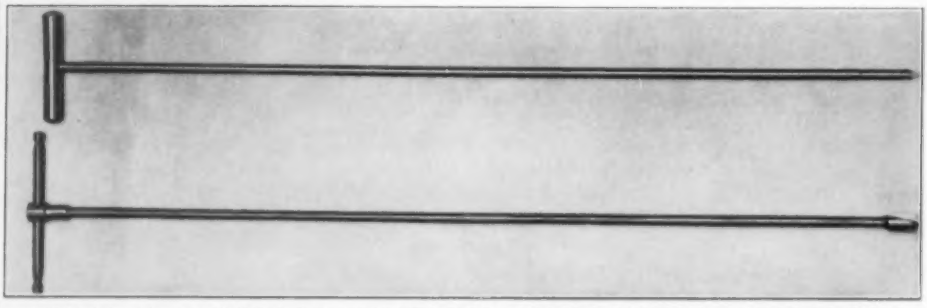
The reamer comes in 3 diameters: ⅜, 7/16, and ½ inch—all are 23 inches in length.



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specific treatment for arthritic joints

intra-articular/intrasynovial/intrabursal instillation

□ **good to excellent response in a vast majority of patients**—"Low doses...provided rapid and effective relief...in almost all of the 157 patients treated..."[1] "...appeared to be superior as an intra-articular injectable substance to anything hitherto available."[2]

□ **provides sustained, long lasting benefits**—In 28 out of 34 patients,"...complete relief was provided by a single injection...the relief lasting for an average of more than 2.5 months."[2]

□ **rapid relief of pain, swelling, and improved range of motion**—"Pain was relieved in 3 or 4 days, or less..."[3] "...marked improvement in range of motion occurred in all of these patients."[3] "...more potent, milligram for milligram, than other injectable corticosteroids."[4]

□ **undesirable side reactions outstandingly rare**—"...appears to be a safe, potent, and effective preparation..."[5] "...tolerated as well as or better than hydrocortisone or prednisolone."[6]

Dosage: usual doses—2.5 to 5.0 mg. for smaller joints; 5.0 to 15.0 mg. for larger joints. *Side Effects:* outstandingly rare; although systemic effects do not ordinarily occur with Kenalog Parenteral when the proper techniques and dosages are used, careful clinical supervision is advisable for all patients receiving steroid therapy. *Contraindications:* infections in or near joints—e.g., gonococcal or tuberculous arthritis. *Supply:* a sterile aqueous suspension in 5 cc. vials, each cc. providing 10 mg. triamcinolone acetonide. *References:* 1. Sperling, I. L.: Clinical Research Notes vol. 3, No. 1 (Jan.) 1960. 2. Steinberg, C. L.: op. cit. 3. Urist, M. R.: op. cit. 4. Meltzer, L. E.: op. cit. 5. Schwartz, S.: op. cit. 6. Felts, W. R.: op. cit.

Among 363 patients treated with Kenalog Parenteral, 315 (86.7%) obtained complete relief or were markedly improved.[1-6]

new **Kenalog Parenteral**

Squibb Triamcinolone Acetonide Aqueous Suspension

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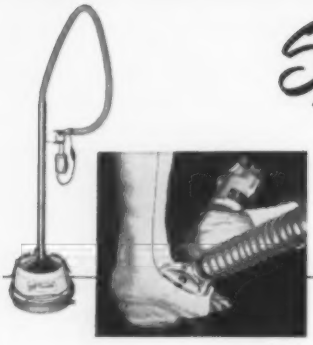
Squibb Quality—the Priceless Ingredient

"KENALOG" IS A SQUIBB TRADEMARK.



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The Stryker Cast Cutter is universally accepted today as the most modern method for quick safe cast removal. The PlasterVac, when attached to the Cast Cutter, instantly picks up all dust and dirt as the cast is cut.

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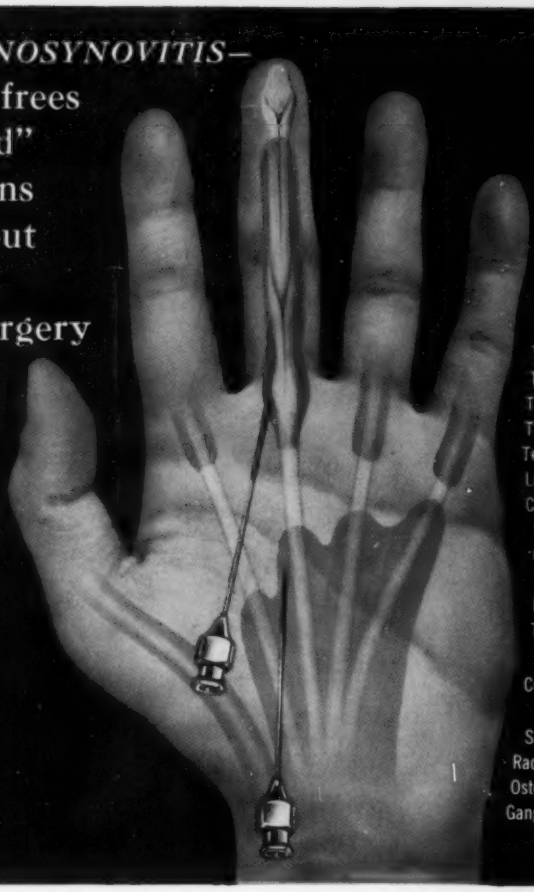
(Prednisolone *tertiary*-butylacetate, Merck)

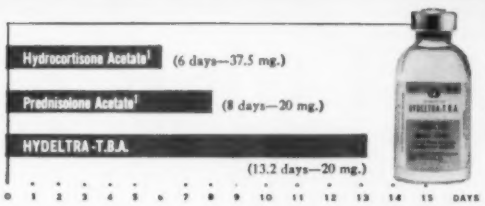
for relief that lasts—longer

in *TENOSYNOVITIS*—
often frees
"locked"
tendons
without
need
for surgery



Osteoarthritis
Rheumatoid arthritis
Acute gouty arthritis
Bursitis
Tendinitis
Trigger finger
Tenosynovitis
Trigger points
Tennis elbow
Lumbosacral strain
Capsulitis
Frozen shoulder
Coccydynia
Rheumatoid nodules
Fibrositis
Tensor fascia lata syndrome
Collateral ligament strains
Sprains
Radiculitis
Osteochondritis
Ganglia

Anti-inflammatory effect lasts longer than that provided by any other steroid ester


Hydrocortisone Acetate[1] (6 days—37.5 mg.)
Prednisolone Acetate[1] (8 days—20 mg.)
HYDELTRA-T.B.A.
(13.2 days—20 mg.)
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 DAYS

Dosage: the usual intra-articular, intra-bursal or soft tissue dose ranges from 20 to 30 mg. depending on location and extent of pathology.

Supplied: Suspension 'HYDELTRA'-T.B.A.—20 mg./cc. of prednisolone *tertiary*-butylacetate, in 5-cc. vials.

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DIVISION OF MERCK & CO., INC.
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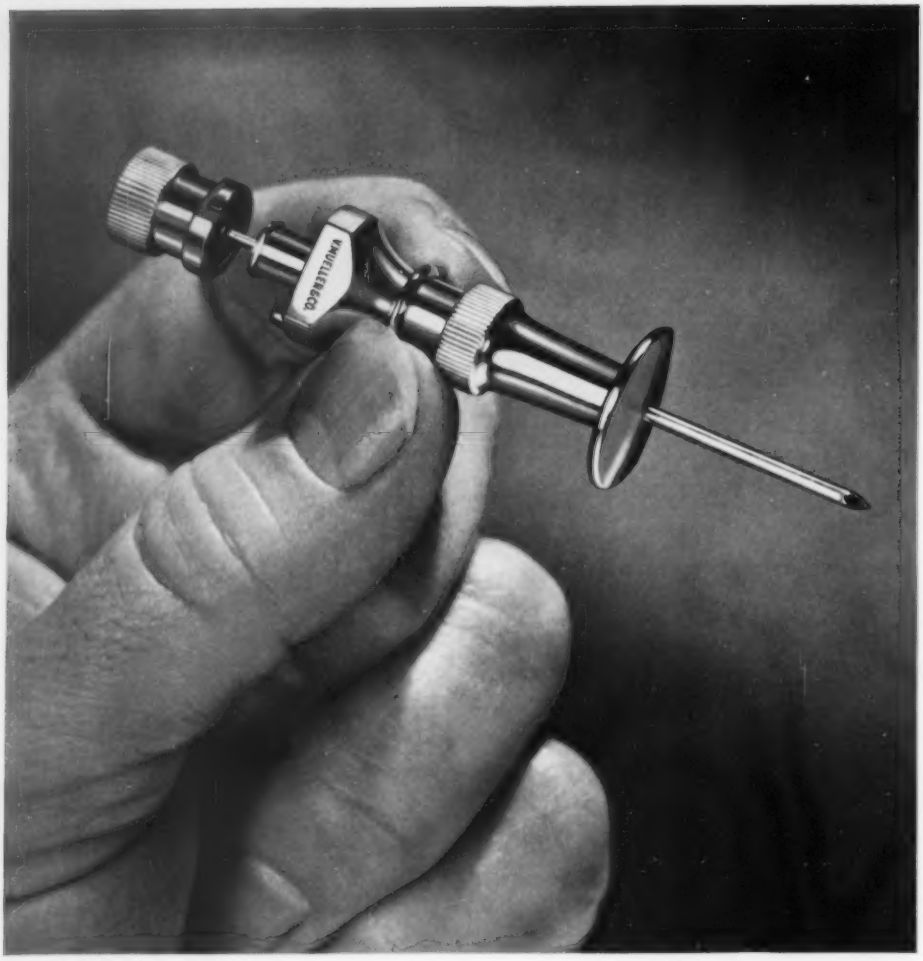

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6-60

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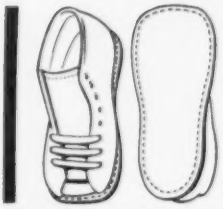
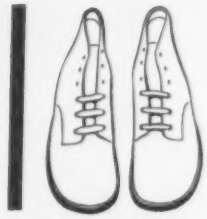


Pre-Walker, reverse or straight last

that have

Pigeon-Toe, for mild supination

earned the



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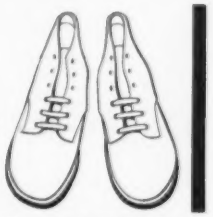
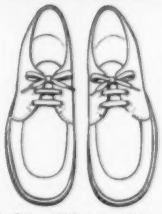


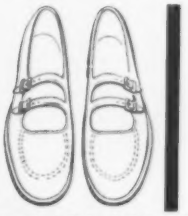
Prona-Pedic, for extreme supination

CorecTreds



CorecTred, for mild pronation

and



Thomas Heel Extended Counter

Pedics by **Edwards**

For over 60 years **Edwards** has sought to develop a *complete line* of fully adaptable, quality shoes, at sensible prices, to meet doctors' special prescription requirements. Using tested lasts from clinic-approved patterns, **Edwards** experienced craftsmen developed CorecTreds and Pedics, the most versatile shoes available, sold by conscientious retailers trained in prescription fitting.

Whatever your prescription requirements, examine and advise CorecTreds and Pedics by **Edwards**!

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THE SHOE FOR CHILDREN
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Now, receive Edwards new, complete Pedic Kit designed especially for you. It contains:

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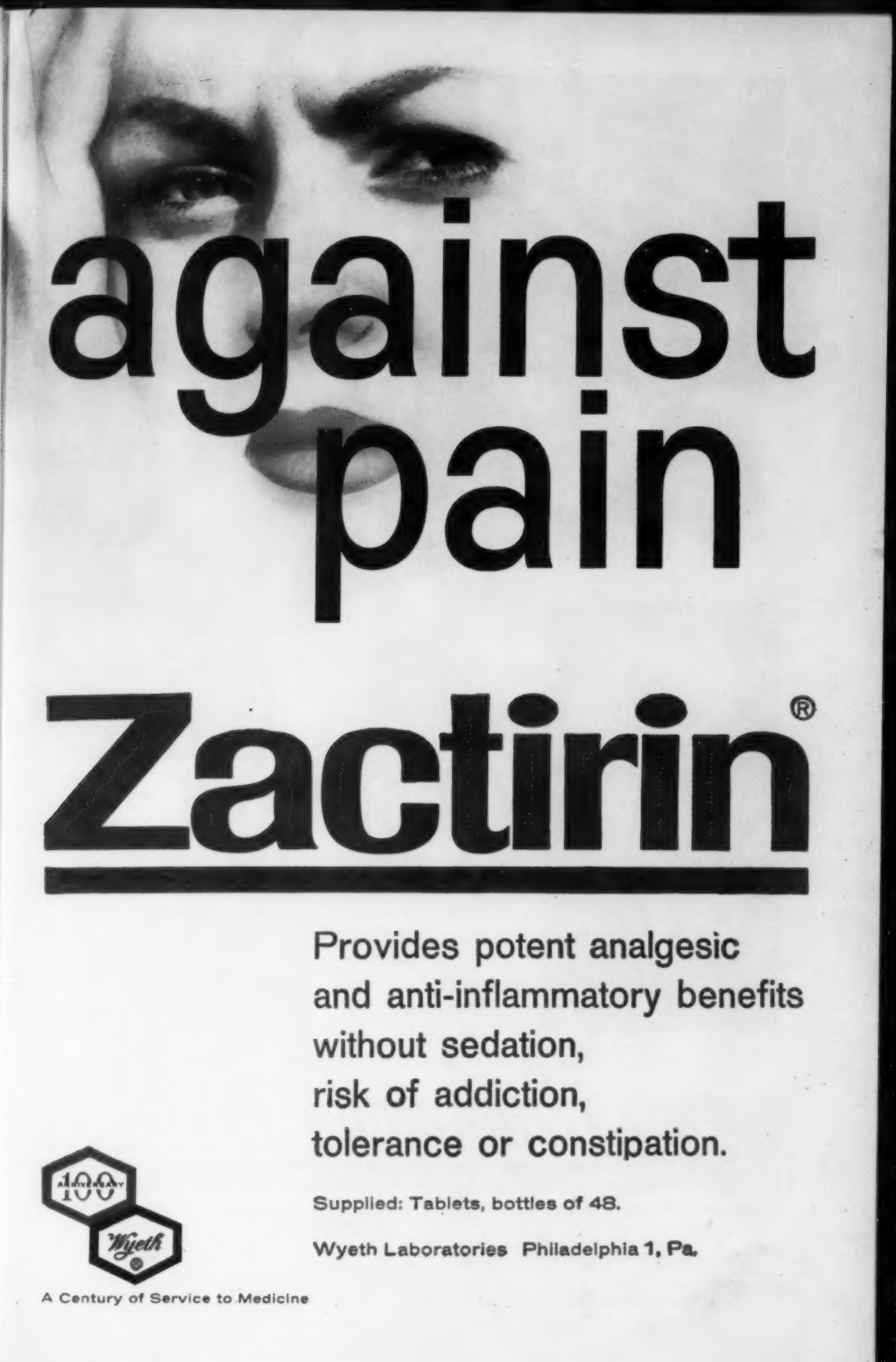
Simply Detach This Portion of Advertisement, Mail With Your Personal Letterhead to Edwards Shoes, 314 N. 12th St., Phila. 7, Pa.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Zactirin®
Provides potent analgesic
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without sedation,
risk of addiction,
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A Century of Service to Medicine

SMO HIP PROSTHESES

AUSTIN-MOORE

Head Diameter	Shaft Length
1⅝"	5"
1¾"	5"
1⅞"	5½"
2"	6"
2-1/16"	6"
2⅛"	6"

THOMPSON

Head Diameter	Shaft Length
1⅝"	5½"
1¾"	5½"
1⅞"	5½"
2"	5½"
2-1/16"	5½"
2⅛"	5½"

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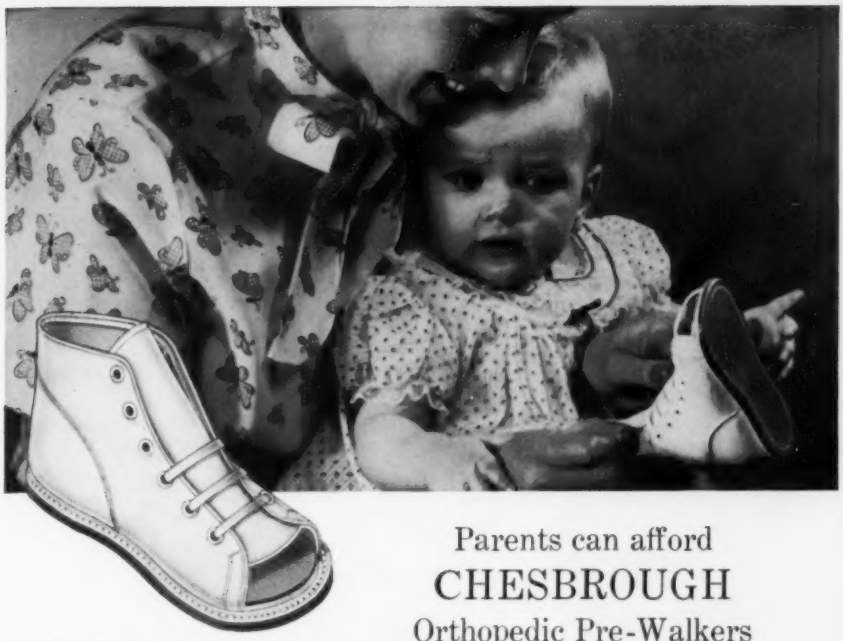

Parents can afford CHESBROUGH Orthopedic Pre-Walkers

No. 1400 OPEN TOE
Straight-line symmetrical last, firm heel, no back seam. Adaptable to Denis Browne Splints.

No. 1300 CLOSED TOE
Lace-to-toe design permits snug, gentle fit. Perfectly smooth inside.

No. 1700 CLUBFOOT, OPEN TOE
Special outflare last, sturdy instep strap to stabilize heel.

Louis C. Weld, President of G. W. Chesbrough Co. . . . "My own personal experience led to the development of the new Chesbrough Shoe."

Here are Orthopedic infants' shoes that meet rigid requirements of corrective footwear yet fit the family budget.

High-quality workmanship and fine leathers with orthopedically correct lasts are combined in Chesbrough Shoes to sell at a moderate price.

Any parent whose child requires orthopedic correction knows the expense is great, as frequent purchase of new shoes is required.

This problem was brought home to Chesbrough's president, Louis C. Weld, three years ago, when a child in his own family needed such a shoe. Recognizing the need for orthopedic shoes at an economical price, Chesbrough Company put their 60 years of shoemaking experience to work and Chesbrough Corrective Pre-Walker Shoes were born.

All shoes in unlined white elk, sizes 000 to 4, narrow and wide. Available in full pairs, split pairs or single shoes (no extra charge for half pairs).

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A fitting service you can prescribe, with confidence . . .

Your Truform anatomical supports dealer is an expertly trained and skilled technician.

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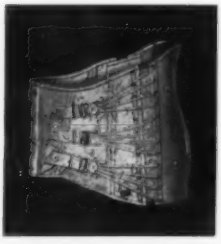

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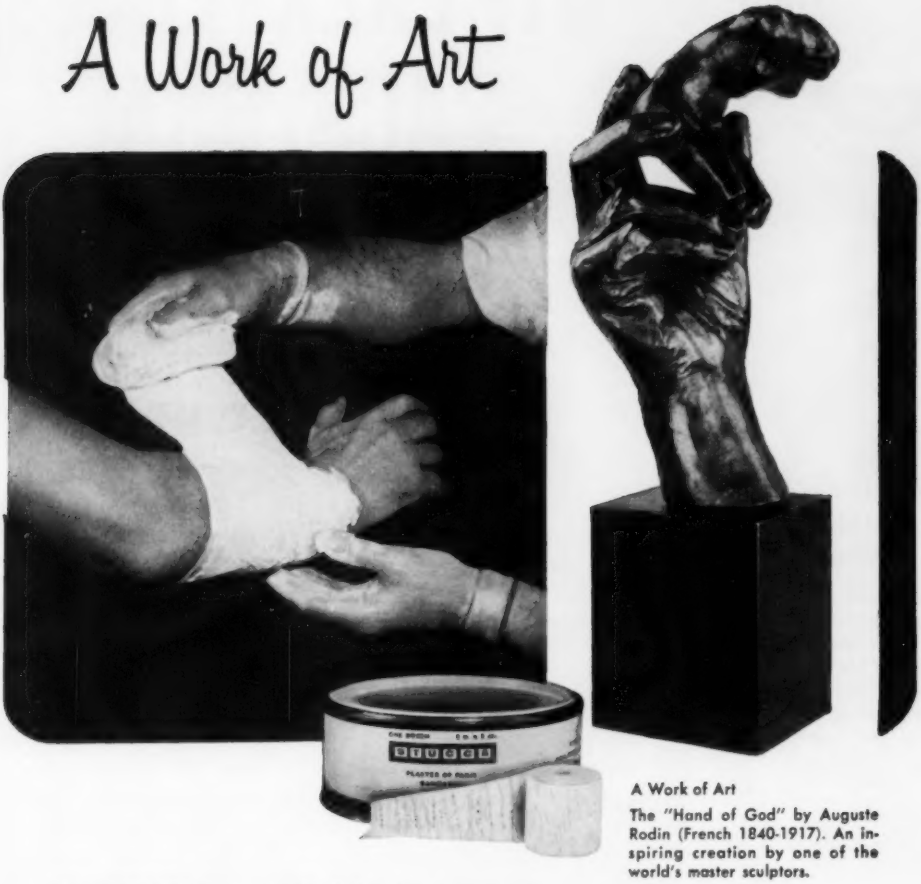

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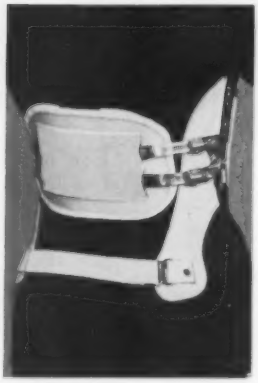

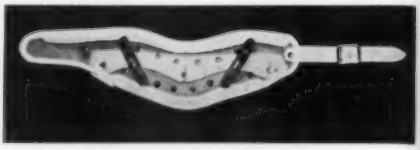

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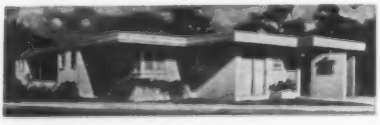

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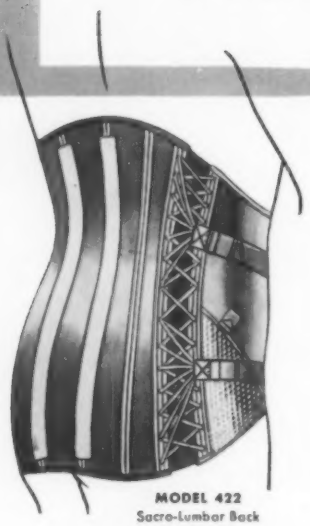

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1. Adelman, M.N., et al.: J.A.M.A. *169*:5 (Jan. 3) 1959.

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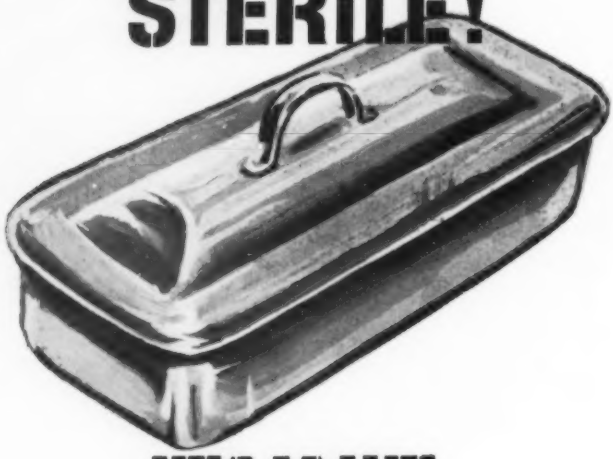

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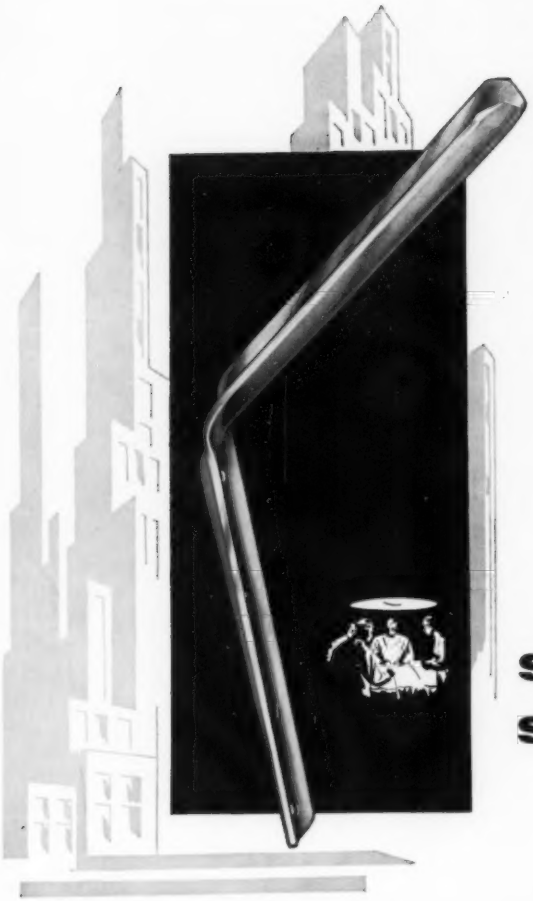

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"The replacement prosthesis has a very definite place in surgery of the arthritic hip, especially when the head of the femur is essentially gone or inadequate."[9]

In the past 5 or 6 years the well-established criteria for replacement arthroplasty have been notably expanded to include refractory rheumatoid- and osteoarthritides. Femoral head replacements in these conditions offer great promise in those individuals where crippling is severe, or where drug therapy does not seem to facilitate ambulation. Clinical results of numerous hip arthroplasties in rheumatoid- and osteoarthritides have proved highly gratifying both immediately and after lengthy follow-up. [2-8]

Aside from shorter hospital stays, hip arthroplasty offers additional advantages in that secondary surgical procedures are seldom required, nursing care is eased, and the economic burden of the patient is thus reduced. Moreover, prosthetic surgery of the hip affords earlier and more complete ambulation, freedom from pain and limp in most cases, and by providing an almost "normal" hip, enables these patients quickly to return to an active, useful life. [1, 11, 13-20]

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REFERENCES: 1. Beegel, P. M.: J. Maine M. A. 49:86, 1958. 2. Moore, A. T.: J. Bone & Joint Surg. 39A:811, 1957. 3. Symposium on Femoral Head Replacement Prostheses, J. Bone & Joint Surg. 38A:407, 1956. 4. Stinchfield, F. E. et al.: J. Bone & Joint Surg. 39A:1043, 1957. 5. Thompson, F. R.: J. Bone & Joint Surg. 36A:489, 1954. 6. Moore, A. T.: South. M. J. 45:1015, 1952. 7. Gaenslen, F. G.: Wisconsin Med. J. 59:181, 1960. 8. Moore, A. T.: In Acad. Orthop. Surg. Instructional Course Lectures, vol. 16, C. V. Mosby, 1959, p. 309. 9. Aufranc, O. E.: Med. Forum, Mod. Med. 27:33, 1959. 10. Thompson, F. R.: In Acad. Orthop. Surg. Instructional Course Lectures, vol. 16, C. V. Mosby, 1959, p. 299. 11. Savastano, A. A., et al.: Rhode Island Med. J. 41:425, 1958. 12. Day, P. L. and Hinchey, J. J.: Clin. Orthop., No. 6, p. 27, 1955. 13. Breck, L. W.: Southwestern Med. 35:207, 1954. 14. Hart, G. M.: J. Lancet 78:369, 1958. 15. Peterson, L. T. and Raad, M. A.: Med. Ann. D. C. 24:233, 1955. 16. Carlander, L. W.: Minnesota Med. 42:566, 1959. 17. Mahoney, J. W. et al.: Am. J. Surg. 95:577, 1958. 18. Barker W. E.: Manitoba Med. Rev. 38:673, 1958. 19. Matchett, F.: Rocky Mountain Med. J. 56:47, 1959. 20. Bascom, J. et al.: J.A.M.A. 169:1863, 1959.

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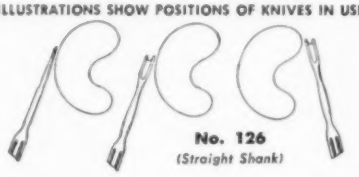

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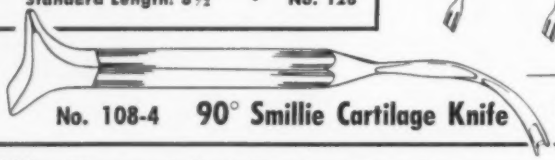

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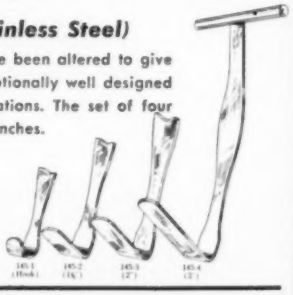

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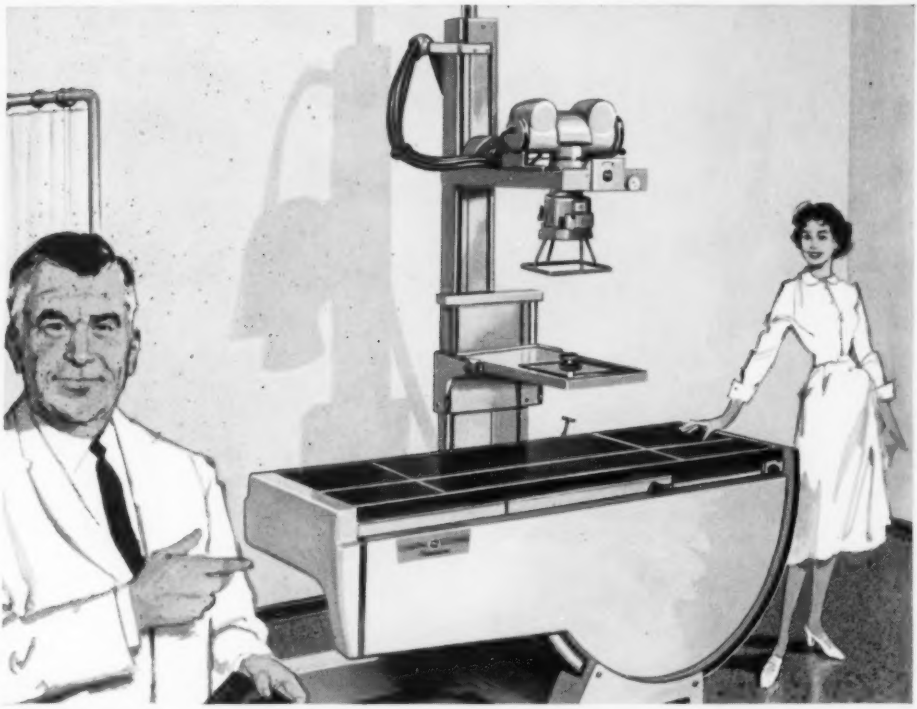

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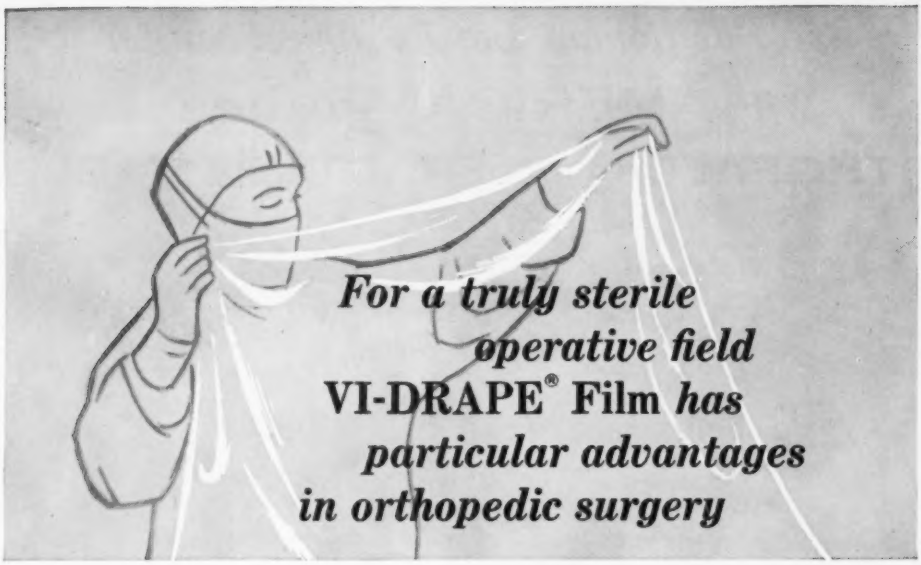

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The Journal of
Bone and Joint Surgery
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Stabilization of the Cervical Spine by Anterior Fusion*

BY R. W. BAILEY, M.D., AND C. E. BADGLEY, M.D., ANN ARBOR, MICHIGAN

From the Department of Surgery, Section of Orthopaedic Surgery, The University of Michigan Medical Center, Ann Arbor

Anterior fusion of the cervical spine has been performed in certain instances at University Hospital since 1952 and has proved itself of distinct value in effecting stabilization. The value of dorsal, or posterior, spine fusion is well accepted, and the indications for posterior fusion are quite clear. The purpose of this report is to indicate instances in which anterior fusion may be superior to or an attractive alternative to posterior fusion.

In 1952, while serving as a visiting professor of surgery at The University of Michigan, Dr. LeRoy C. Abbott suggested an anterior approach to the cervical spine and the possibility of doing an anterior fusion in a woman, twenty-one years old, who had an extensive lytic lesion involving the fourth and fifth cervical vertebrae. The ease of the approach and the ready achievement of stability in this individual stimulated the application of this approach and technique to other lesions of the cervical spine, including fracture-dislocations and destructive lesions of the cervical vertebrae caused by neoplasms and infections. This procedure has also been used to achieve stability in "spontaneous" dislocations of the cervical spine after extensive previous laminectomy and in lesions of the cervical intervertebral discs.

More recently, with the knowledge that anterior fusion may be performed at a subsequent time to restore stability, the neurosurgeon has been allowed greater freedom in the performance of laminectomy in instances of tumors of the cervical spinal cord and other lesions for which decompressive laminectomy is performed.

FRACTURE-DISLOCATIONS OF THE CERVICAL SPINE

Protection of the spinal cord in the treatment of fractures, dislocations, and fracture-dislocations of the cervical spine stands as the prime objective. Rogers [16] summarized the important points by stating that (1) in treatment, the cord must be protected at all times; (2) reduction must be complete or pain and recurrence of deformity will follow; and (3) fixation must be adequate or recurrence of deformity will follow. To attain the objectives, spine fusion in fracture-dislocations of the cervical spine has been advocated by many authors. Our own experiences since

* Read in part at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 29, 1959.

FIG. 1-A FIG. 1-B FIG. 1-C

Figs. 1-A through 1-E: Case A. Note the alteration in height of the intervertebral disc at the time of dislocation and with recurrence of the deformity. We believe that derangement of an intervertebral disc may be associated with faulty repair, leading to recurrence of the deformity.

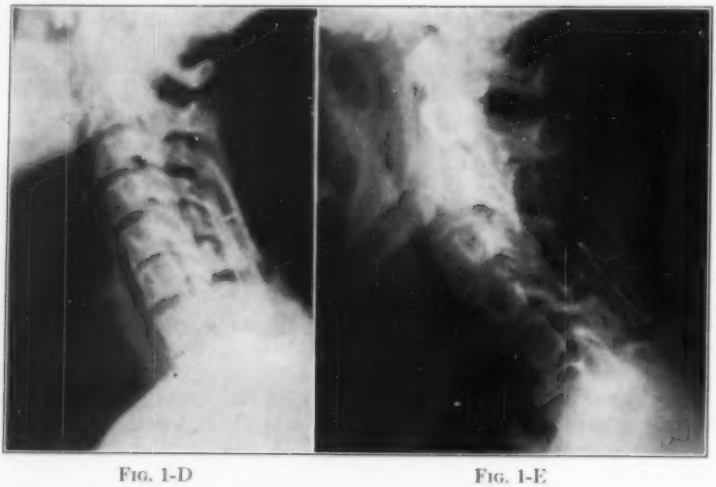

FIG. 1-D FIG. 1-E

Twenty years after posterior fusion of the cervical spine (Fig. 1-E). The patient leads a normal life as an engineering professor. Neck motion is slightly restricted.

1928 have been confirmed by the reports of Cone and Turner, Gallie, and Rogers. These authors pointed out that in many cases fractures of the pedicle or articular processes do not heal well. As the patient assumes the upright attitude, the deformity may slowly or quickly recur in a few days or over a period of months. At a later time there is also a distinct possibility that with trauma of even a minor degree, redisplacement may occur, with drastic neurological consequences.

DISRUPTION OF THE INTERVERTEBRAL DISC

Disruption of the intervertebral disc is a significant pathological feature of these recurring deformities that has been verified by the anterior approach and is worth emphasizing. To our knowledge, this observation of disruption has not been previously described, although it was first predicated by one of us (C.E.B.) in 1939. The sequence of pathological events in the more common type of forward-

FIG. 2

Case B. Initial injury in 1941, with incomplete reduction of fracture-dislocation, with dislocation of the sixth on the seventh cervical vertebra. In 1947, there was minor trauma with immediate quadriplegia. Note the marked decrease in the height of the intervertebral disc space and the fracture through the spur at the anterosuperior aspect of the seventh cervical v

flexion injury is as follows: (1) tearing of the ligamentum nuchae, interspinous ligaments, ligamentum flavum, and, occasionally, the dura; (2) fracture or fracture-dislocation of the articular processes, with or without fractures in the laminae; (3) rupture of the posterior longitudinal ligament; and (4) disruption of the intervertebral disc.

Through the anterior approach to the cervical spine, disruption of the intervertebral disc was observed in a number of patients reported in this study. The annulus was found torn from its attachment to the cephalad vertebral body, which was dislocated anteriorly, with the result that the annulus and nucleus pulposus remained attached to the caudal vertebra. The nucleus itself was found to be fragmented and, in late cases, to be mucoid. We believe that injury to the intervertebral disc is probably the most important factor contributing to early and late displacement. A similarity exists between inadequate healing of the intervertebral disc and the lack of repair observed with a tear of the semilunar cartilage of the knee. The following cases serve to illustrate this concept:

CASE A. On Christmas Eve, 1939, an engineering professor, forty-five years old, was tussling with two of his nephews. While one boy had a full Nelson grip on him, the other jumped on his neck. The patient fell to the floor, complaining of severe pain in his neck and profound weakness in all four extremities. The neurological complaints and findings were transient. Roentgenographic examination revealed a fracture and dislocation of the fifth cervical vertebra on the sixth (Fig. 1-A). The patient was placed in a Stryker frame, and skull-tongs traction was applied. Reduction was accomplished in three days (Fig. 1-B), and three weeks later, traction was discontinued and the patient's neck was immobilized in a Taylor brace with an attached Forrester collar.

Three months after injury, the patient returned for evaluation; he had no complaints and was carrying on his usual teaching activities while wearing the brace. Roentgenograms revealed complete recurrence (Fig. 1-C) of the dislocation. Especially to be noted in Figures 1-A and 1-C is the marked alteration in the interspace between the fifth and sixth cervical vertebrae. It was from these

FIG. 3-A

FIG. 3-B

Fig. 3-A: Case C. In March 1958, the patient sustained a so-called whiplash injury with rapidly developing ascending quadriplegia seventy-two hours after injury. Note the separation of the spinous processes between the fifth and the sixth cervical vertebrae with forward riding of the facets at that level.

Fig. 3-B: January 1959. The patient had persistent pain and discomfort since injury, with abnormal motion between the fifth and the sixth cervical vertebrae, as shown on dynamic flexion-extension roentgenograms.

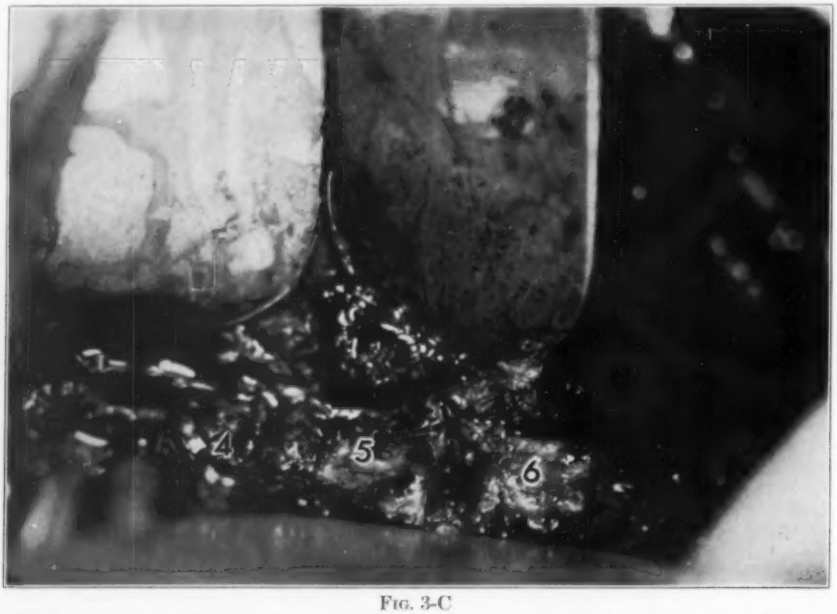

FIG. 3-C

With cervical traction applied, the intervertebral disc space opens to a greater extent between the fifth and sixth cervical than between the fourth and fifth cervical vertebrae. As traction was alternately increased and decreased, the nucleus pulposus would pop forward like a pea from a pod, indicating inadequate repair of injury to the intervertebral disc.

FIG. 3-D
Four months after anterior fusion of the cervical spine.

roentgenograms that the disc injury was first suspected. The redisplacement was corrected with traction, and cervical fusion (Fig. 2-D) was performed from the second cervical to the first thoracic vertebra, using a technique similar to that described by Rogers [8]. Twenty years later the patient had no complaints concerning his neck, but there was some restriction of cervical-spine motion (Fig. 1-E).

Faulty repair after injury to the intervertebral disc may result in poor healing. As a result, with trauma of even a minor degree, vertebral displacement may occur years later, with disastrous neurological consequences.

CASE B. In 1941, a man, forty-nine years old, sustained a fracture and dislocation of the sixth cervical vertebra on the seventh in a fall. Reduction of the dislocation was accomplished in skull-tongs traction, and, three weeks later, the patient was placed in a Minerva jacket (the roentgenograms made at that time were, unfortunately, destroyed). Some recurrence of dislocation occurred, but operative reduction and fixation were refused by the patient. As time went by, an area of calcification developed between the anterior aspects of the sixth and seventh cervical vertebrae, but spontaneous fusion between the vertebral bodies did not occur across the intervertebral disc space.

Six years later, in 1947, the patient sat down on a folding chair that collapsed and he became quadriplegic immediately. He did not specifically recall striking his head. Roentgenographic examination revealed a break through the area of calcification that had developed between the anterior aspects of the sixth and seventh cervical vertebrae. Of particular interest was the absence of any roentgenographic evidence of spontaneous fusion between the vertebral bodies (Fig. 2). He was placed in traction on a Stryker frame. Some months later he died from an ascending urinary-tract infection complicating a cord bladder.

The constant increase of automobile collisions has stimulated a great deal of medical and legal interest in the so-called whiplash injury. The following report may shed some light on the sequence of events and the pathology in injuries of this type:

CASE C. On March 7, 1958, a man, twenty-one years old, sustained an injury to his neck when the car in which he was riding was struck from behind while it was stopped at a traffic signal. The impact was sufficient to break the seat free from its attachments. The patient did not recall striking his head, but had severe pain in his neck, and, within an hour, he had pain in the suboccipital area as well. He was taken to the hospital and placed in head-halter traction.

On examination, tenderness was present in the posterior cervical area, associated with muscle spasm and limitation of cervical-spine motion in all directions. Neurological examination was normal. Roentgenograms revealed no gross dislocation. There was, however, a definite separation of the fifth and sixth cervical spinous processes, which we have found to be a frequent sign indicating a disruption of the ligamentum nuchae and interspinous ligaments with or without a partial subluxation of the interarticular facets (Fig. 3-A).

The patient was placed in bed with head-halter traction, which offered symptomatic relief. Thirty-six hours after injury, he awoke with a feeling of numbness and weakness in the lower extremities. This progressed and within four hours involved the trunk and arms. Hyporeflexia in the triceps and both lower extremities was present, with hypalgesia below the level of the sixth dermatome.

Immediate laminectomy of the fourth through the sixth cervical vertebrae was performed. On inspection, the cord exhibited slight swelling, but beyond this was normal. Anterior to the cord, possible intervertebral disc extrusion was searched for, but none was found.

Two weeks after laminectomy, sensation and strength reappeared in the arms, and, about two weeks later, recovery in the lower extremities appeared. This was followed soon by re-establishment of normal bowel and bladder function. Six weeks later, the patient was discharged from the hospital wearing a Forrester collar.

Because of severe neck pain whenever he attempted to discontinue use of the brace, the patient was referred to University Hospital in January 1959. Forward displacement of the fifth on the sixth cervical vertebra was demonstrated on lateral roentgenograms of the cervical spine in flexion and extension (Fig. 3-B). Spine fusion was performed through an anterior approach. The area from the fourth to the seventh cervical vertebrae was exposed. The disc between the fifth and sixth cervical vertebrae was soft to palpation and bulged anteriorly. The anterior longitudinal ligament was dissected off the anterior aspects of the fifth and sixth cervical vertebrae, and a small portion of the annulus was excised from the disc spaces between the fourth and fifth and fifth and sixth cervical vertebrae. Operation was performed with the patient in skull traction. As traction was increased, the space between the fifth and sixth cervical vertebrae opened much more widely than did the normal disc spaces between the fourth and fifth and sixth and seventh cervical vertebrae. As traction was alternately increased and decreased, the nucleus pulposus between the fifth and sixth cervical vertebrae would pop in and out through the surgical defect in the annulus, indicating faulty repair at this disc space (Fig. 3-C). The disc between the fifth and sixth cervical vertebrae was removed. The nucleus pulposus was fragmented and was mucoid and stringy compared with that between the fourth and fifth cervical vertebrae, which was also removed preparatory to fusion. A trough was cut along the anterior aspects of the fourth, fifth, and sixth cervical vertebrae, and an iliac graft was mortised into position (Fig. 3-D).

The significance of the pathological findings is evident and confirms the original hypothesis of Badgley.

In many similar injuries it has been observed by us that as traction is applied during reduction of a cervical dislocation or fracture-dislocation, all the intervertebral disc spaces widen as increasing amounts of weight are applied. At the level of the dislocation, the widening of the interspace is greater than at the other spaces, and the amount of widening depends on the degree of derangement of the intervertebral disc.

The value of posterior spine fusion in the treatment of certain fracture-dislocations of the cervical spine from the occiput to the first thoracic vertebra has been accepted at the University Hospital since 1929. In our experience, no other form of treatment (casts or braces) has consistently served to prevent recurrence of the dislocation. In some instances, the fracture-dislocation may not be reduced by skull-tongs traction because of locking of the interarticular facets. Here, operative reduction of the dislocation is advised, with removal of a portion of the facet if necessary. At the same time, posterior fusion is performed by a technique very similar to that described by Rogers [16].

In certain instances, posterior fusion of the spine is technically difficult and hazardous, and anterior fusion is preferable. Some of the circumstances under which anterior fusion has proved to be of distinct value in the treatment of fracture-dislocations of the cervical spine are as follows:

1. For stabilization of the spine after laminectomy. Cervical fractures, dislocations, and fracture-dislocations have been the combined responsibility of the neurosurgeon and orthopaedic surgeon at University Hospital. If a neurological lesion exists for which laminectomy is indicated, this is performed by the neuro-

surgeon with the orthopaedic surgeon present, and, usually, at the time that laminectomy is completed the orthopaedic surgeon performs cervical fusion, using a cancellous-bone graft placed laterally along the denuded laminae and an encircling wire extending from the spinous process above to that below the laminectomy. However, this combined procedure is not always possible, especially when the dura has been left open, when the laminectomy has been so extensive that major portions of the facets have been removed, or when the laminectomy has been protracted or the patient's condition is precarious. Frequently, during laminectomy, the dentate ligaments are divided, and the cord is mobilized to allow visualization anterior to the cord. Under such circumstances, anterior fusion performed at a later time is especially valuable.

2. For stabilization of fracture-dislocations that are associated with severe maxillofacial injuries. This combination of injuries may be a potential or actual cause of severe compromise of respiratory function. Under these circumstances the anterior route offers an easier and safer way of maintaining an airway during surgery. The operation is performed with the patient supine rather than prone, which is of considerable help to the anesthesiologist. Furthermore, if, at the completion of operation, any disturbance of respiration is present or threatening, a tracheotomy may readily and easily be performed.

3. For the stabilization of the old fracture-dislocation with instability of the spine and quadriplegia. Such instability (Case 2) threatens further damage to the remaining cervical nerve roots. The preservation of all functioning nerve roots for the rehabilitation of these quadriplegics is most important, as emphasized in the current work of Nickel, in which the flexor-hinge hand is formed by late reconstructive surgery. If further injury occurs to an unstable cervical spine, additional neurological damage may result in the patient's death, or the very important previously spared nerve roots may be destroyed.

4. For the stabilization of fracture-dislocations of the cervical spine associated with osteo-arthritis. In one patient, roentgenographic examination revealed distinct osteo-arthritic changes along the posterior aspects of the vertebral bodies at the site of dislocation. The remote, although definite, possibility that, with the passage of time, disturbances of cord or nerve-root function might occur secondary to increasing osteo-arthritic lipping at the level of the fracture-dislocation led to the selection of anterior fusion of the cervical spine. By use of this technique no impediment to future laminectomy and decompression was introduced.

As experience has been gained through use of anterior fusion, application of this method has increased. The relative ease with which the cervical vertebrae can be reached, and the simplicity with which the site to be fused can be roentgenographically identified have led to greater employment of this method. When fusion is indicated for fracture-dislocations that have been reduced between the second cervical and first thoracic vertebrae, the anterior route is an attractive alternative to fusion through the posterior route. However, anterior fusion is not indicated if reduction has not been attained by skull traction, and open reduction through the posterior approach is necessary.

OPERATION

The operative approach and technique described here is used for the forward-flexion type of fracture-dislocation; it is subject to minor alterations as the specific pathological problem demands. This approach is not new and has long been employed in other surgical problems, such as the draining of retropharyngeal abscesses and the exposure of the upper portion of the esophagus. Other authors [2, 15, 20] have reported on their experiences with this approach in treatment of lesions of the cervical intervertebral discs. The route most frequently employed lies just anterior to the sternocleidomastoid muscle.

The operation is performed with the patient on a Stryker frame or Foster bed

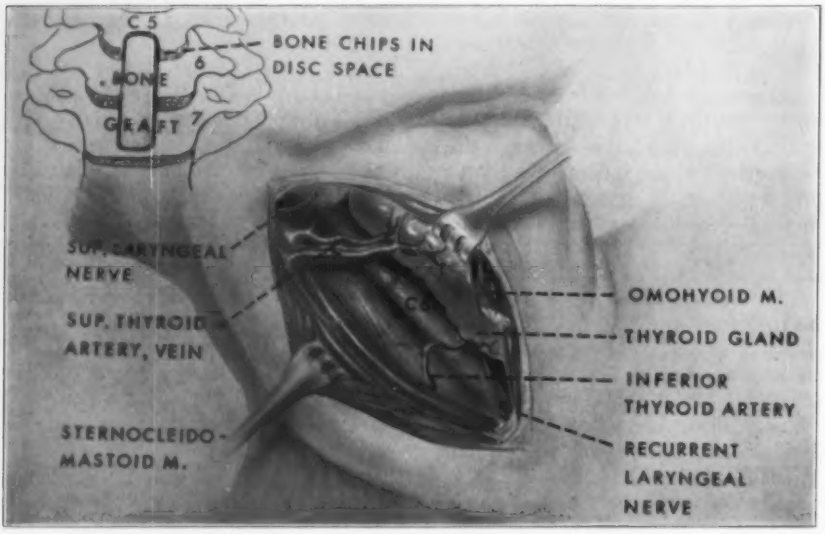

FIG. 4

Anterolateral approach to the cervical spine. The inset indicates the method of fusion with an iliac-bone graft mortised into a carefully prepared bed and cancellous chips gently packed into the disc spaces.

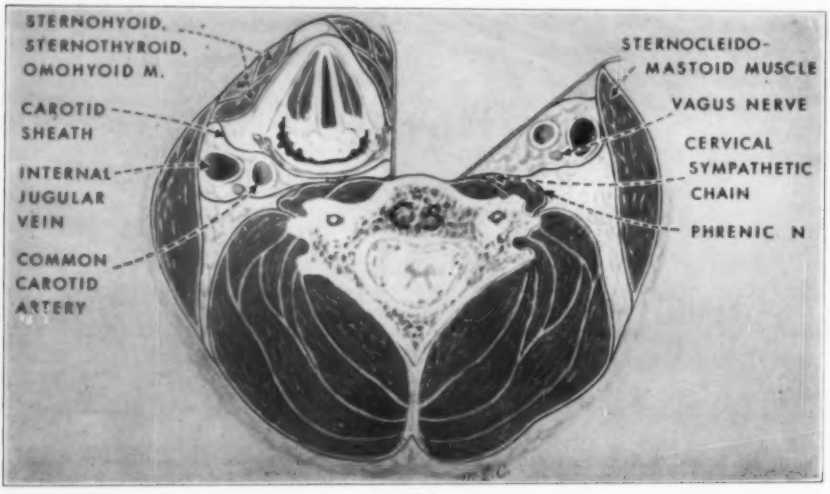

FIG. 5

Cross section showing the anterior neck structures retracted to the left, and the carotid sheath and its contents to the right. Incision through the prevertebral fascia at the exact mid-line with dissection of the longus colli muscles close to bone avoids damage to cervical sympathetic nerves.

after the fracture-dislocation has been reduced with the use of skull-tongs traction. Accurate reduction is an essential prerequisite to anterior fusion. If the fracture-dislocation cannot be reduced (because of locked interarticular facets, for example) posterior fusion is preferred because this permits reduction to be achieved under direct vision, with excision of a part of an articular facet if necessary.

In all instances, endotracheal anesthesia was employed, and intubation was achieved through the oral route, with care being taken not to extend the neck excessively. In patients with severe maxillofacial injuries it may be necessary to

insert the endotracheal tube through the nose. The presence of the tube within the trachea serves as an additional aid in identification of the trachea during the operation.

Proper positioning of the patient is important. A folded towel is placed beneath the interscapular region to maintain the cervical spine in moderate extension. The patient's head is rotated about 15 degrees to the left because approach from the right is preferred.

The incision is made on the right side, beginning two fingerbreadths distal to the mastoid process and extending along the anterior border of the sternocleidomastoid muscle to just above the sternum (Fig. 4). The carotid tubercle can usually be palpated and serves to identify the sixth cervical vertebra. The platysma and anterior layer of the cervical fascia are incised in the same direction as the skin and subcutaneous tissues. The sternocleidomastoid muscle is gently retracted laterally, exposing the carotid sheath and its contents. The pulsating common carotid artery is quite distinct, and care should be taken to avoid traction on this structure. The omohyoid muscle is identified crossing the lower portion of the wound obliquely. It may be gently freed and retracted distally or transected. The pretracheal layer of the cervical fascia is to be incised next, and, here, caution is advised. Crossing superficial to the fascia from medial to lateral in the mid-portion of the wound lies the middle thyroid vein. In most instances, it is the only major vessel that must be divided and ligated during the entire operation. Once this has been accomplished, a small longitudinal incision is made through the pretracheal fascia of sufficient size to allow the surgeon to insert a finger into the retropharyngeal area. Here there is a very loose areolar connective tissue, through which, with gentle persistence, the finger may dissect proximally to touch the base of the skull and distally to the superior mediastinum. As the incision through the pretracheal fascia is extended proximally and distally, the superior thyroid artery and vein and the superior laryngeal nerve are anticipated. In most instances, they can be gently retracted proximally. To reach the interspace between the second and third cervical vertebrae, division of the superior thyroid artery and vein may be necessary. Similarly, to reach the lower cervical vertebrae and cervicothoracic junction it may be necessary to divide the inferior thyroid artery and vein, but this has not been required in this series. The close proximity of the recurrent laryngeal nerve to the inferior thyroid vessels demands special care to avoid injury to it.

The retropharyngeal space exhibits a remarkably loose arrangement of areolar tissue that facilitates cephalad and caudad probing as well as lateral retraction of the anteriorly situated structures. The presence of the endotracheal tube allows easy identification of the trachea by palpation. The trachea, esophagus, anterior ("strap") muscles of the neck, and thyroid gland are gently retracted to the left. The prevertebral fascia is now seen as a glistening firm layer covering the longus colli muscles and the anterior aspects of the cervical vertebrae (Fig. 5).

Orientation as to the vertebrae to be exposed may be aided by palpation, but should be substantiated roentgenographically before the prevertebral fascia is incised. The carotid tubercle can be palpated, identifying the sixth cervical vertebra. Furthermore, the base of the skull may be palpated, and the vertebrae counted down from the ringlike anterior portion of the first cervical vertebra. To ensure that the proper site for exposure and fusion is selected after identification by palpation, a drill is inserted as a marker in one of the vertebral bodies, and a lateral roentgenogram is made.

The prevertebral fascia is incised in a longitudinal direction at the exact mid-line. Heavy silk sutures are placed in the fascia to facilitate retraction and later to aid in closure of the fascia that acts as a strong retainer for the thrust bone graft. As the prevertebral fascia is mobilized from the anterior surfaces of the vertebral bodies, modest bleeding is encountered from the bone and is controlled with electrocautery or bone wax. The site of the dislocation may be

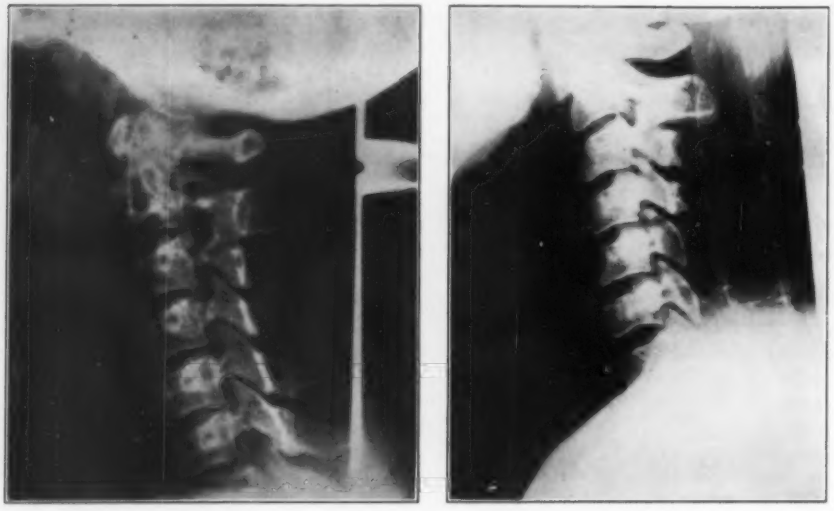

FIG. 6-A FIG. 6-B

Fig. 6-A: Case 7, B. N. (Table I). On January 28, 1958, a nurse, thirty-seven years old, was seen two hours after injury; there was progressive quadriplegia. The roentgenogram was made with the patient's head stabilized on a litter. A fracture-dislocation, with dislocation of the sixth on the seventh cervical vertebra was observed.

Fig. 6-B: Skull-tongs traction was applied. The patient was placed on a Foster bed, and emergency laminectomy of the fifth, sixth, and seventh cervical and the first thoracic vertebrae was performed. The roentgenogram made on the third postoperative day reveals satisfactory reduction of the dislocation.

obvious if one is aware of the softening and bulging of the intervertebral disc, as already described.

The vertebrae to be fused are identified, and a trough is cut in the anterior aspect of the vertebral bodies about one-half of an inch in width and three-sixteenths of an inch in depth. The use of an electric dental saw or drill is an aid here and is less traumatic than the use of an osteotome and a mallet. The trough is cut into the full vertical height of the vertebrae. The intervertebral discs are cleaned with a rongeur to a depth of three-sixteenths of an inch, and the cartilaginous plates on the inferior and superior aspects of the vertebral bodies to be fused are removed.

While the cervical area is being exposed, an iliac-bone graft is obtained. Chips of cancellous iliac bone are gently packed into the cleaned intervertebral disc spaces. The graft is trimmed to fit and mortised into the trough that has been made in the full vertical height of the vertebra. It is essential to pack chips of cancellous bone into the disc space because it is a well known fact of bone-graft surgery that grafts that bridge defects between two osseous structures commonly fail to become part of the host in the region where they are surrounded only by soft tissue (Fig. 4, inset).

The neck has been in moderate extension thus far, but, at this point in the procedure, the cervical extension is decreased by raising of the traction. This wedges the graft more securely in its trough. The graft must not project further anteriorly than the normal anterior surface of the vertical bodies, lest, later on, it behave as a retropharyngeal mass and give rise to dysphagia.

When it is assured that the graft is in proper position, the sutures previously placed in the prevertebral fascia are tied. This fascia acts as an excellent retinaculum to maintain the graft in its bed. A large drain is placed in the retropharyngeal space and brought out through the lower portion of the operative incision. The wound is closed in layers with interrupted sutures, and a light dressing is applied.

FIG. 6-C

FIG. 6-D

Fig. 6-C: Two months after anterior fusion of the cervical spine. The operative route was just posterior to the carotid sheath. Site of fusion was at the fourth, fifth, sixth, and seventh cervical, and the first thoracic vertebrae.

Fig. 6-D: November 1958, ten months after fusion. There was marked improvement in neurological status. The patient was doing regular nursing duty at the time of writing.

After operation, the patient is maintained in traction on a Stryker frame or Foster bed. The drain is advanced in twenty-four hours and removed forty-eight hours after operation. Traction is maintained for six weeks. The patient is then allowed to get up with his neck immobilized in a Taylor back brace with an attached Forrester collar. These are worn until fusion is complete—usually from four to six months.

In one patient in this series on whom cervicothoracic fusion was performed, the approach was made from the patient's left side just posterior to the carotid sheath and its contents. By this route, access to the first and second thoracic vertebrae was easily gained, although subsequent experience has proved that equally easy access to the cervicothoracic area can be achieved from the right side without potential danger to the thoracic duct.

APPLICATION OF ANTERIOR FUSION OF THE CERVICAL SPINE FOR FRACTURE-DISLOCATION OF THE CERVICAL SPINE

The following case reports illustrate some fracture-dislocations in which anterior fusion of the cervical spine was found to be of real value.

CASE 1. B. N. (Case 7, Table I), a nurse, thirty-seven years old, was admitted to the Wayne County General Hospital on January 25, 1958, with the complaint of neck pain and progressive weakness about one hour after an automobile accident in which she had been thrown out of the right front seat onto the street. After the accident she had been picked up, placed in another car, and driven to the hospital. *En route*, numbness and weakness of her legs developed.

While she was being studied in the emergency room, weakness in all four extremities developed. Sensation was lost below the level of the first thoracic dermatome. The deep-tendon responses in both lower extremities were absent but remained present in the upper extremities. There was weakness of the intrinsic muscles of both hands and of the wrist extensors and triceps muscles bilaterally. Respiration was solely diaphragmatic. Motion of the cervical spine was restricted in all directions because of pain, and there was marked tenderness along the posterior mid-line at the cervicothoracic junction.

Roentgenograms revealed fracture-dislocation with anterior subluxation of the sixth cervical vertebra on the seventh and fracture of the seventh cervical vertebra. Lumbar-spine puncture demonstrated complete block to the cerebrospinal-fluid flow. Skull-tongs traction was applied, and the patient was placed on a Foster bed (Fig. 6-A).

Cervicothoracic laminectomy was carried out promptly including the fifth, sixth, and seventh

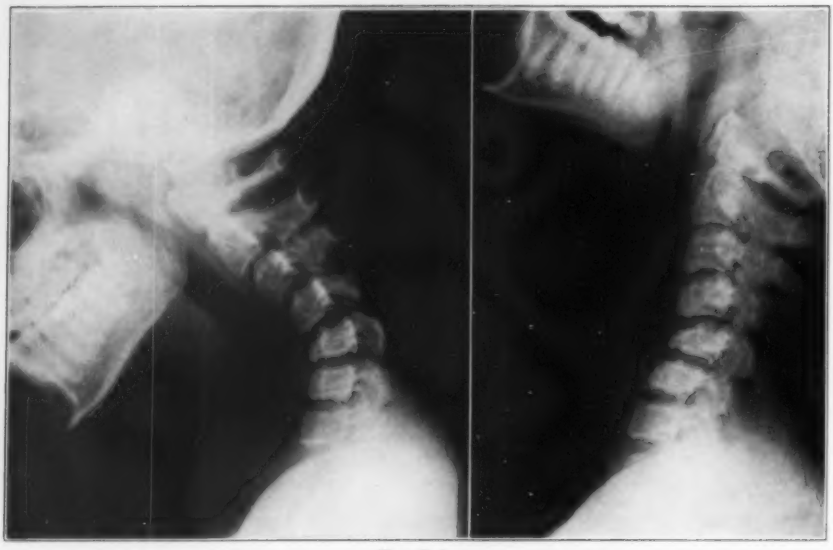

FIG. 7-A

Case 3. L. J. (Table I), a young man, seventeen years old, sustained a diving injury in August 1955. One month later he had an extensive laminectomy. Ten months later he was seen with severe spastic quadriplegia. Mobility was present at the site of the previous fracture-dislocation, with dislocation of the fourth on the fifth cervical vertebra. Rehabilitation efforts were deferred because of instability in the cervical area.

FIG. 7-B

FIG. 7-C

Fig. 7-B: Anterior fusion of the cervical spine at the fourth, fifth, and sixth cervical vertebrae. The graft is shown in the anterior trough one week after prophylactic tracheotomy because of disturbed respiratory function.

Fig. 7-C: Two years after anterior fusion. Note the maturity of the bone bridge.

cervical and the first thoracic vertebrae. The spinal cord was slightly swollen but of normal color and without evidence of contusion or ecchymosis. The dentate ligaments were divided in this area, and exploration anterior to the cord was carried out. No bone displacement or encroachment on the cord was demonstrated (Fig. 6-B). The dura was not closed.

Gradual improvement in neurological status occurred during the next month. On February 25, 1958, anterior fusion from the fourth cervical to the first thoracic vertebra was performed. On this occasion, the approach was made from the left side just posterior to the carotid sheath (Fig. 6-C).

FIG. 8-A

FIG. 8-B

Fig. 8-A: Case 6. D. R. (Table I), a woman, thirty-four years old, admitted after an automobile accident on January 25, 1958, in acute respiratory distress. Note the bilateral temporomandibular-joint dislocation and the hyperextension type of dislocation of the second on the third cervical vertebra. Aeration was further compromised by fractures of the maxillae and body of the mandible.

Fig. 8-B: The dislocation was reduced with the patient in skull-tongs traction on a Foster bed.

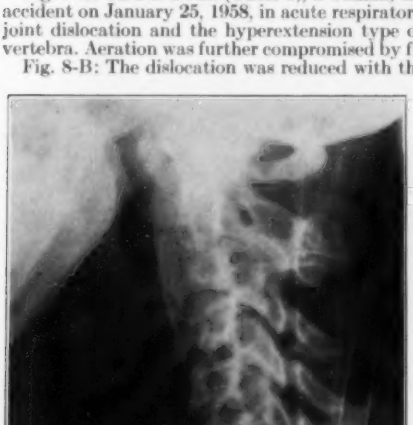

FIG. 8-C

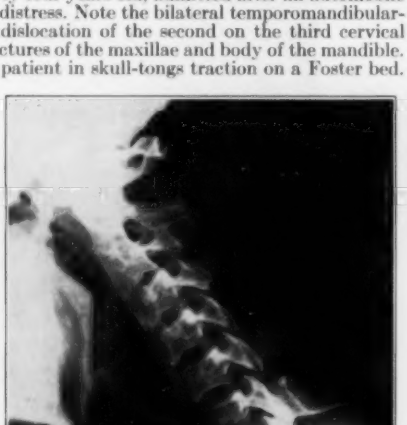

FIG. 8-D

Fig. 8-C: Anterior fusion of the cervical spine was performed on February 25, 1958. Roentgenograms made one month later show fusion extending from the second to the fourth cervical vertebrae.

Fig. 8-D: Ten months after fusion, showing solid union from the second to the fourth cervical vertebrae.

Skull-tongs traction was discontinued six weeks later, and a program of active rehabilitation was commenced in June. The patient has shown a progressive return of muscle strength that is still continuing. Marked muscle weakness persists below the right knee, but the strength of her arms is almost normal. There has been complete recovery of bowel and bladder function. She has resumed part-time nursing responsibilities (Fig. 6-D).

CASE 2.* L. J. (Case 3, Table I), a young man, seventeen years old, was admitted to Ranchos Los Amigos Hospital, Downey, California, in February 1956, with posttraumatic quadriplegia.

* Courtesy of Dr. Vernon L. Nickel and Ranchos Los Amigos Hospital, Downey, California.

In August 1955, he had sustained a fracture-dislocation with dislocation of the fourth on the fifth cervical vertebra and a compression fracture of the fifth cervical vertebra. Some deep sensation was preserved in the lower extremities. An extensive laminectomy had been performed a short time after injury, with little improvement in the paralysis. Gradually, spasticity in both the arms and the legs had developed. On entry to Ranchos Los Amigos Hospital, the patient complained of some neck pain and, on flexion-extension roentgenograms, demonstrated gross instability at the interspace between the fourth and fifth cervical vertebrae (Fig. 7-A). Attempts at rehabilitation were believed to be very hazardous because of the instability of his neck and the risk of additional damage to the spinal cord were he to fall. Anterior spine fusion was performed from the fourth to the sixth cervical vertebrae on May 26, 1956 (Fig. 7-B). Immobilization was continued in a plaster bed for three months, after which intensive further rehabilitation measures were carried out.

The patient has made excellent progress in subsequent rehabilitation efforts. An automatic bladder developed, no longer requiring a catheter. He lives at home, attends regular school, and gets about in a wheelchair with minimum assistance (Fig. 7-C).

FIG. 8-E

Photograph made one year after operation. These photographs, made with the jaws closed, indicate the malocclusion with loss of contact of the incisors; the overbite; and how the jaw motion is restricted by the proximity of the mandible (upper black mark) and superior border of the thyroid cartilage (lower black mark).

Preservation of the remaining functioning nerve roots was essential for this patient's rehabilitation. With the muscles innervated by the spared nerve roots, this boy is able to be self-sufficient.

The value of the anterior approach in cases of combined fracture-dislocation with maxillofacial injuries is demonstrated in the following case report. It is worthy of note that the interspace between the second and third cervical vertebrae was accessible through the anterior approach.

CASE 3. D. R. (Case 6, Table I), a woman thirty-four years old, was admitted to the hospital on January 25, 1958, from the scene of an accident involving the car that she was driving. She was in acute respiratory distress, blood was issuing from her mouth, and there were a number of facial lacerations. There were obvious deformities of both mandibles, the nose, and malar area. Emergency tracheotomy was performed immediately, resolving the acute respiratory condition. Apart from the patient's facial injuries and her complaint of pain in the upper cervical area, no other abnormalities were noted. Neurological examination was within normal limits.

One year before injury the patient had complained of neck pain, and roentgenograms of the

cervical spine demonstrated a congenital block vertebra involving the second and third cervical segments. Roentgenograms at the time of injury showed dislocations of both temporomandibular joints, bilateral fractures near the angles of the mandibles, and fracture of the left maxilla. There was a hyperextension type of fracture-dislocation of the cervical spine, with the body of the second cervical vertebra displaced posteriorly on the third cervical vertebra (Fig. 8-A). Skull-tongs traction was applied, and, within a few days, complete reduction of the dislocation was accomplished (Fig. 8-B). After five days the tracheotomy tube was removed.

Anterior fusion of the cervical spine was selected because of the severe deformity of the mandibles that made the administration of anesthesia in the prone position hazardous. Anterior fusion from the second to the fourth cervical vertebra was performed on February 25, 1958 (Fig. 8-C). The patient was kept on a Foster frame in skull-tongs traction from the time of admission through April 7, 1958, when she was allowed to get up, wearing a Taylor back brace with a Forrester collar; this apparatus was discontinued in July 1958.

Figures 8-D and 8-E demonstrate the patient's course and status at the time of writing. She has no complaints apart from those related to the appearance and function of her jaw. Reconstructive surgery is planned in the near future.

Case 4. J. C. (Case 5, Table I), a man, fifty-two years old, was transferred to Wayne County General Hospital on February 19, 1957, with complaints of a so-called broken neck and weakness in all four extremities. On January 5, 1957, he slipped and fell, striking his head on a dresser. He was unconscious for about one hour and when he regained consciousness, he was unable to move both arms and his right leg. On arrival at a nearby hospital, in addition to the paralysis, he complained of severe pain with numbness and tingling in both arms. After roentgenograms were made of the cervical spine, the patient was placed in a Minerva jacket and discharged home. The weakness in his legs improved, and strength in his right arm returned. He had no bowel or bladder complaints after the injury, but was unable to gain an erection. Because of persistent severe burning pain in his neck and both shoulders and arms, he went to a second hospital, where his cast was removed, and new roentgenograms were made. Immediate hospitalization was advised, and he was transferred to Wayne County General Hospital.

Examination on admission revealed severe restriction of neck motion in all directions because of pain. Moderate tenderness was present in the mid-line in the mid-cervical area. Distinct muscle weakness was present in both upper extremities, but was more marked on the left. The greatest weakness was found in the triceps and biceps muscles. The triceps and biceps tendon responses were diminished bilaterally. The responses of the Achilles and patellar tendons were bilaterally equal and active. Babinski's sign was not present. The abdominal and cremasteric reflexes were absent on the right. No weakness was noted in the lower extremities. There was hypesthesia over the entire forearm on the left, on the medial aspect ot the forearm and hand on the right, in the saddle area, and over the dorsal surfaces of both feet.

Roentgenograms revealed a fracture-dislocation with anterior displacement of the fifth cervical vertebra on the sixth segment. There were considerable hypertrophic arthritic changes involving the fifth, sixth, and seventh cervical vertebrae (Fig. 9-A).

At the time of admission to the hospital, skull-tongs traction was applied, and the patient was placed on a Foster bed. Lumbar-spine puncture was performed, and no evidence of obstruction of the cerebrospinal-fluid flow was found. The spinal fluid was clear; it contained no cells on microscopic examination. The cerebrospinal-fluid protein was elevated to 118 milligrams per 100 milliliters.

The dislocation was promptly reduced with cervical traction (Fig. 9-B). The neurosurgical consultant advised against laminectomy in view of the progressive improvement in neurological status since the patient's injury and the lack of any evidence of obstruction to the flow of the spinal fluid. Spine fusion to prevent recurrence of the dislocation was advised. Because of the rather marked degenerative arthritic changes along the posterior aspects of the vertebral bodies and the possibility of additional lipping at a future time requiring laminectomy, the anterior route was selected.

On March 5, 1957, anterior cervical fusion was performed with the patient in traction and on a Foster bed. After operation, traction was maintained for six weeks. The patient was then allowed to be up with a Taylor brace with an attached Forrester collar. This apparatus was worn through June 1957 (Fig. 9-C).

At the time of writing, there has been marked improvement in the neurological picture although some weakness of the grip of the left hand persists along with weakness of the elbow muscles. Hypesthesia of the palmar aspect of the second and third fingers of the left hand remains. The patient has no other complaints and is able to work and to lead a normal life (Fig. 9-D).

SPONTANEOUS DISLOCATIONS AFTER EXTENSIVE PRIOR LAMINECTOMY FOR LESIONS OTHER THAN FRACTURE-DISLOCATIONS

Although not commonly recognized or reported in the literature, a factor that may be responsible for late or spontaneous dislocation of the cervical spine is extensive laminectomy. Occasionally, it may be necessary to remove extensive

FIG. 9-A

FIG. 9-B

Fig. 9-A: Case 5. J. C. (Table I), a man, fifty-two years old. Roentgenogram made in March 1957, one month after injury, demonstrates fracture-dislocation, with dislocation of the fifth on the sixth cervical vertebra. Previous reduction and immobilization in a plaster Minerva jacket were unsatisfactory in preventing redisplacement. Note the decrease in the height of the fifth and sixth and sixth and seventh cervical intervertebral disc spaces, with posterior osteophytic proliferation.

Fig. 9-B: Four days after skull-tongs traction was started, with the patient on a Foster bed. The deformity was corrected with ease.

FIG. 9-C

FIG. 9-D

Fig. 9-C: Three months after anterior fusion of the cervical spine at the fifth and sixth cervical vertebrae with evidence of considerable healing in the bone graft and bridging of bone across the intervertebral disc space anteriorly.

Fig. 9-D: One and one-half years after anterior fusion of the cervical spine. The bone graft appears well incorporated into the host area. Further bridging across the intervertebral disc space is noted.

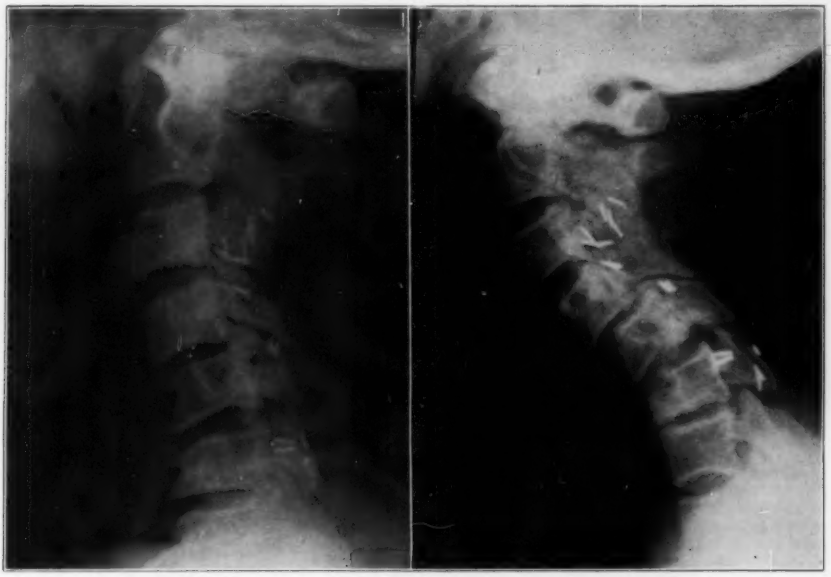

FIG. 10-A

Case 2. E. M. (Table I). In November 1955, this man, fifty-four years old, had neck pain and progressive weakness in all four extremities. In 1931, a large cervical meningioma had been removed because of similar complaints, with complete improvement. The symptoms gradually began to recur twenty-four years later. Lateral roentgenograms in flexion and extension demonstrate anterior subluxation of the fourth on the fifth cervical vertebra.

FIG. 10-B

FIG. 10-C

Fig. 10-B: One month after anterior fusion of the fourth and fifth cervical vertebrae. The patient was in skull-tongs traction and on a Stryker frame.

Fig. 10-C: Three years after anterior fusion of the cervical spine. The intervertebral disc space between the fourth and fifth cervical vertebrae appears completely bridged by bone.

portions of the posterior neural arch to gain exposure for removal of tumors of the spinal cord, ruptures of the intervertebral disc, and cysts of the spinal cord.

FIG. 11-A

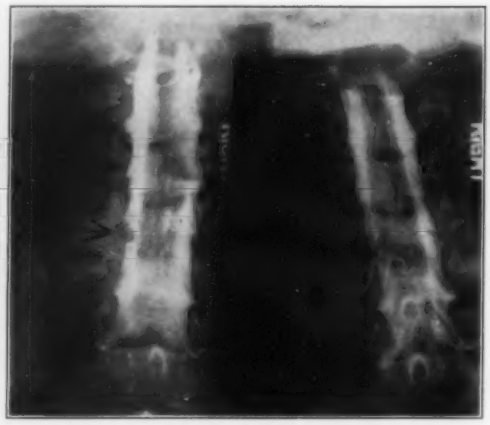

FIG. 11-B

Fig. 11-A: Case 4. K. D. (Table I), a man, forty-two years old, had a rear-end-collision injury, with neck and arm pain. No abnormality was noted in May 1952.

Fig. 11-B: Myelogram, made in February 1953. There was a defect due to herniation of the nucleus pulposus. Hemilaminectomy on the left was done, with removal of a ruptured disc between the fifth and the sixth cervical vertebrae. The symptoms completely disappeared.

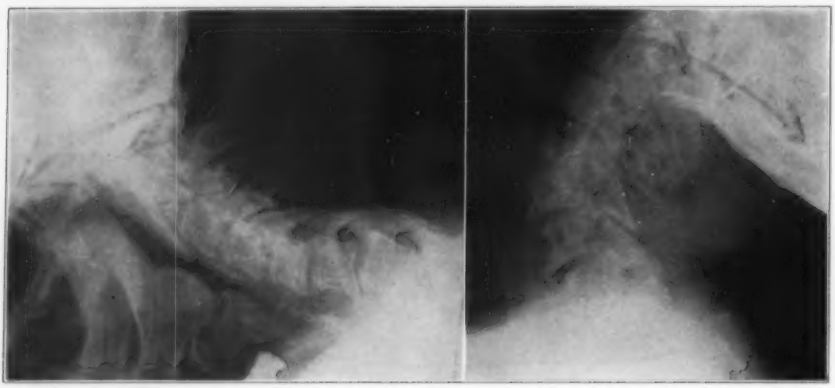

FIG. 11-C

Distinct subluxation of the sixth on the seventh cervical vertebra was clearly demonstrable both on the roentgenogram and on direct vision at the time of anterior fusion.

The surgeon must be aware that with laminectomy of this magnitude, especially when portions of the interarticular facets are removed, forward dislocation may occur either shortly or many years after the surgical procedure, with disastrous neurological consequences.

In one patient included in this study, displacement gradually occurred twenty-five years after the removal of a meningioma of the cervical cord with production of a progressive quadriplegia caused by traction on the cord. The neurological symptoms were corrected by reduction of the dislocation and anterior fusion. Two other patients with similar conditions were seen and treated successfully in the same fashion.

In view of these complications, any patient with an extensive laminectomy, especially if portions of the facets have been removed, should be observed carefully for a spontaneous dislocation. If clinical or roentgenographic evidence of dislocation appears, as shown by dynamic flexion-extension roentgenograms, fu-

sion must be considered to prevent damage to the cord. Anterior fusion offers obvious advantages under such circumstances.

CASE 5. E. M. (Case 2, Table I), an automobile worker, fifty-four years old, entered University Hospital in November 1955, with a complaint of weakness in all four extremities that had slowly progressed over the previous year and had recently become severely disabling, making walking and standing impossible. In 1931, twenty-five years previously, he had been treated for similar complaints. An extensive laminectomy that included the third, fourth, fifth, and sixth cervical vertebrae had been performed, and a large meningioma of the cervical spinal cord was removed. He had had almost complete recovery from this operation and had led a completely normal life until early in 1955.

FIG. 11-D

One and a half years after anterior fusion at the sixth and seventh cervical vertebrae for "spontaneous" dislocation after extensive previous laminectomy. The patient was completely symptom free.

On admission to University Hospital, neurological examination revealed a posterior mid-line healed scar from the first to the seventh cervical vertebrae. Spontaneous fasciculations in the pectoral and deltoid muscles were present bilaterally, as well as marked atrophy and weakness of the right deltoid and triceps and left triceps. Diffuse but marked weakness was present in both hands. Muscle tone in both lower extremities was increased. Position and vibratory senses in both lower extremities were impaired. The deep-tendon responses were increased in both lower extremities, with Babinski's sign present bilaterally. Sensation was normal above the level of the sixth cervical dermatome. Over the sixth cervical dermatome the patient was unable to discriminate sharp from dull stimuli, and hypalgesia and hypesthesia were noted over the trunk and legs.

Roentgenograms revealed evidence of extensive laminectomy of the third, fourth, fifth, and sixth cervical vertebrae, with anterior displacement of the fourth cervical vertebra on the fifth apparent on flexion-extension roentgenograms (Fig. 10-A). The neurosurgical consultant believed that the symptoms and signs were not caused by recurrence of the neoplasm but by traction on the cord due to the dislocation.

Skull-tongs traction was applied on November 14, 1955. During the next week, marked subjective and objective improvement in the neurological picture occurred. By December 1955, the patient's paralysis had almost completely disappeared. During this time, serial roentgenograms showed that reduction of the dislocation was accomplished. Anterior fusion between the fourth and fifth cervical vertebrae was carried out on December 14, 1955 (Fig. 10-B).

Six months after operation, the patient returned to work and has been fully active since. He was last seen in June 1960, when his only residual disability was slight persistent weakness below the left knee (Fig. 10-C).

FIG. 12-A

FIG. 12-B

Fig. 12-A: Case 1. L. S. (Table I). Roentgenogram made in April 1952, showing a lytic lesion that had destroyed the bodies of the fourth and fifth cervical vertebrae. A prominent retropharyngeal mass can be seen. There is destruction of portions of the pedicles and facets of the fourth and fifth cervical vertebrae. Biopsy revealed an aggressive giant-cell tumor.

Fig. 12-B: July 1952. Since April there had been (1) partial laminectomy of the fourth and fifth cervical vertebrae combined with fixation, using a No. 18 steel wire from the second to the seventh cervical vertebra with a Hibbs-type bone graft; (2) a third course of x-ray therapy; (3) anterior removal of the tumor with anterior decompression of the spinal cord; (4) anterior fusion of the cervical spine, using an iliac-bone thrust graft from the inferior portion of the third cervical into the upper portion of the sixth cervical vertebra.

CASE 6.* K. D. (Case 4, Table I), a man, forty-two years old, had sustained an injury to his neck when the automobile that he was driving was struck from behind in May 1952. After this he complained of pain in the neck and left arm that was refractory to a variety of treatment programs. In February 1953, a left hemilaminectomy was performed by a neurosurgeon, and a ruptured intervertebral disc was removed from the interspace between the fifth and sixth cervical vertebrae, giving prompt relief from complaints (Figs. 11-A and 11-B). Four months later, in May 1953, he again sustained a neck injury in a second automobile collision. Again, conservative measures failed, and, in September 1953, a ruptured intervertebral disc was removed from the interspace between the sixth and seventh cervical vertebrae. After the second operation, pain persisted and responded only to narcotics; these had been used to the point of severe addiction, and the patient began gradually to manifest general deterioration.

Examination in 1956 revealed an asthenic, nervous, pitiful-appearing man of forty-six years. Neck motion was severely restricted in all directions because of pain. Flexion-extension roentgenograms revealed very definite dislocation of the sixth on the seventh cervical vertebra, accentuated by forward flexion (Fig. 11-C).

A trial of immobilization in a Minerva jacket for a period of one month resulted in complete relief of pain and eliminated the need for narcotics almost entirely.

In June 1956, anterior fusion of the cervical spine was performed. At operation there was gross mobility between the sixth and seventh cervical vertebral bodies. With slight flexion of the cervical spine, the body of the sixth vertebra displaced anteriorly on the seventh by almost one-half of its width. After operation immobilization in a Minerva jacket was continued for four months. A Thomas collar was then worn for an additional two months.

The patient resumed work in December 1956, and had remained free of all complaints at the time of writing. Surprisingly enough, he sustained a third injury to his neck in January 1957, when his automobile was again struck from behind. On this occasion he had no subsequent difficulty (Fig. 11-D).

The initial stimulus for anterior decompression of the spinal cord and anterior

* Courtesy of Dr. James D. Brown, Arcadia, Cal ..ia.

FIG. 12-C

FIG. 12-D

Fig. 12-C: December 1952. The status of the anterior thrust graft is satisfactory. There appeared to be additional destruction of the facets of the fourth and fifth cervical vertebrae.

Fig. 12-D: April 1953, one year after institution of treatment. The anterior thrust graft is satisfactory. There is further evidence of tumor activity as observed in further destruction of the facets of the fourth and fifth cervical vertebrae. Six months later the patient gradually became quadriplegic and died. Death was believed to be due to tumor involvement of the spinal cord.

fusion of the cervical spine was given by a patient with an aggressive giant-cell tumor that had completely destroyed the bodies of the fourth and fifth cervical vertebrae. Progressive quadriplegia was overcome by the decompression, and stability was achieved through anterior fusion.

Since 1952, two additional patients with rapidly progressing quadriplegia secondary to destructive lesions in the cervical vertebrae have been treated by anterior decompression and fusion. In one patient, the lesion was caused by tuberculosis, and neurological recovery occurred rapidly during the first two postoperative weeks. In the second patient with osteomyelitis due to *Pseudomonas aeruginosa*, recovery has been slower.

The following case is of historical significance in that the patient had the first known fusion by this approach; her case served as the stimulus to apply this approach to fracture-dislocations in the cervical spine.

CASE 7. L. S. (Case 1, Table I), a woman, twenty-one years old, was first admitted to University Hospital in April 1952 because of pain in her neck and right shoulder due to a lytic lesion of the cervical spine that had destroyed the body of the fourth cervical vertebra completely and that of the fifth cervical vertebra partially. Considerable lysis of the pedicles of these vertebrae was also evident. In December 1950, she had first complained of pain in her shoulder, and, at that time, an area of bone destruction was present in the body of the fourth cervical vertebra. Intensive treatment with x-ray therapy was followed by complete relief of symptoms. No biopsy was obtained prior to this treatment. In December 1951, pain in the neck and left shoulder recurred and, again, a course of x-ray therapy was given without the taking of a biopsy. This time, no improvement in symptoms was experienced, and further consultation was sought. During the three months prior to admission, the patient's pain increased, and paresthesia developed in the left arm; she also lost fifteen pounds of weight.

Examination in April 1952 revealed a chronically ill, asthenic woman with fixed torticollis rotating the chin downward and to the left. No neurological abnormalities were observed in the lower extremities. Diffuse weakness and ease of fatigue were present in the upper extremities. The tendon reflexes of the right biceps and left triceps were absent. Severe restriction of neck motion was present because of pain with moderate tenderness over the spinous processes of the fourth, fifth, and sixth cervical vertebrae.

Roentgenographic examination showed an extensive lytic lesion that had destroyed the entire body of the fourth cervical vertebra and part of the body of the fifth cervical vertebra, with

extensive destruction of the pedicles of these two vertebrae. A large retropharyngeal mass was visible, which displaced the tracheal shadow to the left (Fig. 12-A).

Biopsy of the lesion through a vertical incision just anterior to the right sternocleidomastoid muscle revealed a large mass that was removed incompletely. Histologically, this was a highly cellular neoplasm, consisting of multinucleated giant cells and spindle cells, which was interpreted as an aggressive giant-cell tumor.

On May 2, 1952, a Hibbs cervical fusion was performed through a posterior approach from the second to the seventh cervical vertebrae, utilizing a wire loop that extended from the second to the seventh cervical vertebrae as internal fixation. Skull-tongs traction had been applied before the operation, which was carried out with the patient on a Stryker frame. At this operation, a limited laminectomy of the fourth and fifth cervical vertebrae was performed, leaving the lateral extremities of the laminae and interarticular facets intact. No evidence of tumor was observed in the laminae. The dura and spinal cord appeared normal. The immediate postoperative status was satisfactory, but, within seventy-two hours, a Brown-Séquard syndrome developed, with marked weakness in the right arm and leg. It was anticipated that as edema subsided, the patient's neurological status would improve, During the period from April 29 to May 20, x-ray therapy amounting to a total dosage of 1400 r was given through the thoracic area. Little improvement in the Brown-Séquard syndrome was noted.

On May 26, 1952, anterior decompression of the spinal cord through the anterior approach* was carried out, with the removal of as much of the tumor as possible. The tumor appeared to be limited by the intervertebral disc between the third and fourth cervical vertebrae cephalad and that between the fifth and sixth cervical vertebrae caudad. The posterior longitudinal ligament could not be identified, and the tumor abutted directly on the dura over the anterior aspect of the spinal cord. A thrust bone graft that would extend between the bodies of the third and sixth cervical vertebrae had been contemplated at this time, but this procedure was postponed because of considerable bleeding, which was finally controlled by packing of the space between these vertebrae with gelfoam.

After this operation, the wound healed promptly and, within one month, decided improvement in the Brown-Séquard syndrome was apparent.

On June 13, 1952, a thrust bone graft obtained from the iliac crest was inserted between the bodies of the third and sixth cervical vertebrae. On June 30, after the operative sites were well healed, the skull tongs were removed, and the patient was kept on bedrest wearing a combined Taylor back brace and Forrester collar. The brace was worn constantly except at times of sponge bathing in bed (Fig. 12-B).

In September 1952, the patient began to walk. Apart from slight weakness of dorsiflexion of the right wrist, her neurological status was normal, and she resumed normal life without her brace. She remained asymptomatic until April 1953, when pain in her neck recurred. A progressive quadriplegia developed, and she died at home in July 1953.

The last roentgenograms, obtained in April 1953, revealed solid fusion from the third to the sixth cervical vertebra, with the thrust graft being incorporated, but there was evidence of further destruction of the interarticular facets between the fourth, fifth, and sixth cervical vertebrae (Figs. 12-C and 12-D).

Case 8. J. P. (Case 16, Table I), a man sixty-four years old, was transferred to University Hospital in May 1959 ten days after cervical laminectomy. He arrived in skull-tongs traction that had been applied prior to laminectomy.

Four weeks before admission to University Hospital the patient quite suddenly became aware of some fullness of the abdomen, followed within a day by so much difficulty in using his right leg that he began to use a cane. Over the next three days, progressive weakness in both lower extremities developed to the extent that he was unable to walk without a great deal of assistance. He was taken to the emergency room of a nearby hospital where physical examination revealed an incomplete quadriplegia at the level of the sixth cervical segment of the spinal cord. Roentgenograms revealed a destructive lesion of the sixth cervical vertebra. He was immediately placed in skull-tongs traction, and a myelogram was performed that was interpreted as showing a block to the passage of the dye at the level of the sixth cervical vertebra. A laminectomy was performed that included the fourth through the seventh cervical vertebra, and a portion of the first thoracic vertebra. No abnormalities were encountered. After operation, skull-tongs traction was continued. Gradual improvement in hand strength and return of activity in the lower extremities occurred during the next ten days.

A review of the patient's history suggested that his difficulties probably began in January 1958, when, for no apparent reason, pain developed in the posterior cervicothoracic area that extended toward both shoulders. Roentgenograms made at that time were said to be normal, but since then the patient had noticed some weakness in both hands and difficulty in flexing his neck so that he had pain when he attempted to look down at his feet when standing erect. Although his pain improved for a while with heat and massage, he became so uncomfortable during the summer

* The authors are indebted to Dr. John Orebaugh and Dr. Richard C. Schneider for their aid in this first operation.

FIG. 13-A

FIG. 13-B

Fig. 13-A: Case 16 (Table I). Roentgenogram made in June 1959, of J. P., a man sixty-four years old, two weeks after laminectomy performed for rapidly progressing quadriplegia. Note the extensive destruction of the sixth cervical and the upper portion of the seventh cervical vertebra. Note the prominent retropharyngeal abscess.

Fig. 13-B: Six months after operation. Note that the soft-tissue-abscess shadow is gone. The fusion from the fifth to the seventh cervical vertebrae appears satisfactory.

of 1958 that he was unable to work as a laborer. In November, however, he had returned to work and continued working through January 1959.

In February 1959, a small painless white nodule appeared on the scrotum over the left testicle and began to drain. Cultures of this nodule revealed the presence of the tubercle bacillus, and urine cultures obtained at that time were also positive for tuberculosis. Antimicrobial therapy with isonicotinic acid and para-aminosalicylic acid was instituted. The discharge from his scrotal lesion ceased, and his general condition seemed to be improving until the onset of the paresis.

On admission examination in May 1959 at University Hospital, recent weight loss was evident, although the patient was well developed. The cervical-laminectomy incision was well healed. A mass was palpable in the region of the left epididymis with evidence of a healed sinus tract. Rather marked atrophy of the interossei was present in both hands with diffuse weakness of the hands and wrists. The triceps-tendon reflex was absent on both sides; the biceps-tendon reflex was absent on the right. There was marked weakness of both lower extremities. Babinski's sign was present bilaterally together with hyperreflexia in the lower extremities and absence of the abdominal reflexes. General hypesthesia was present over the trunk and lower extremities but an exact level could not be determined. Vibratory sense was decreased over the lower extremities up to about the level of the hips.

Laboratory studies were within normal limits, except for elevation of the erythrocyte sedimentation rate and a persistent microscopic pyuria. Intravenous pyelograms showed changes consistent with tuberculosis of the left kidney.

Roentgenograms of the cervical spine showed extensive destruction of the body of the sixth cervical vertebra, the intervertebral disc between the sixth and seventh vertebrae, and the superior surface of the seventh cervical segment. A large retropharyngeal soft-tissue shadow was assumed to be an abscess. In addition, there was evidence of the previous laminectomy that included the fifth, sixth, and seventh cervical, and a portion of the first thoracic vertebrae (Fig. 13-A).

In the absence of abnormality of the cord at the time of laminectomy, the disturbance of cord function was interpreted as due to traction on the cord secondary to the instability caused by the destructive lesions in the sixth and seventh cervical vertebrae. Because of the prior laminectomy and the large retropharyngeal mass, which was thought to be protruding posteriorly at the involved disc space, decompression of the abscess, removal of caseous material, and cervical fusion by the anterior route were considered to be the treatment of choice. A large abscess that contained thick yellow material was encountered just beneath the prevertebral fascia. The region of the disc space between the sixth and seventh vertebrae was filled with caseous fibrous material. The bodies of the sixth and seventh vertebrae were largely destroyed. The detritus was removed down to healthy bleeding bone in preparation for the reception of the bone graft. The posterior longitudinal ligament was visualized and appeared to be intact. The intervertebral disc between the fifth and sixth

FIG. 14-A

Case 18. L. J. (Table I). July 1959, two years after a twisting neck injury and one year after laminectomy at which time a ruptured disc was removed between the fifth and sixth cervical vertebrae. Additional injury four months later resulted in recurrence of neck pain and brachialgia. Note the changes between the fifth and sixth cervical vertebrae. There is considerable narrowing of the intervertebral disc space.

FIG. 14-B

December 1959. No complaints. The patient engaged in full activity.

vertebrae also appeared involved and it was removed. An autogenous iliac-bone graft was fashioned and inserted as a thrust graft from the fifth to the seventh cervical vertebra.

Histological studies of the excised tissue suggested caseous tuberculosis, but tubercle bacilli could not be cultured from the retropharyngeal abscess and they were not demonstrated by guinea-pig inoculation.

This patient's postoperative course was characterized by rather dramatic and rapid improvement in motor function in all four extremities with return of normal sensation. After operation he was kept in skull-tongs traction on a Stryker frame for six weeks. Since the traction was discontinued, the patient has continued to be on bedrest with his neck immobilized in a Taylor brace with an attached Forrester collar. At the time of writing antituberculosis treatment is being continued at a sanatorium. Fusion appears to be occurring, and the graft does not appear to have undergone any destruction (Fig. 13-B).

ANTERIOR FUSION OF THE CERVICAL SPINE IN LESIONS OF THE CERVICAL INTERVERTEBRAL DISC

Cloward, Smith and Robinson, and Southwick and Robinson reported on their experiences with removal of the cervical intervertebral disc and fusion through the anterior approach. Cloward's technique differs from that of the other authors because he reported visualization of the cord and roots by means of removal of a core of bone from the vertebral bodies contiguous to the intervertebral disc anteriorly. The technique described by Smith and Robinson and Southwick and Robinson resembles more closely our approach. We have limited the use of anterior disc removal and fusion to two patients with lesions of the intervertebral disc whose cases are described in detail.

There are two reasons why we have not extended the anterior approach to problems of herniation of the cervical intervertebral disc. First, it is our opinion that when the cervical cord or nerve roots are subjected to pressure by herniation of the nucleus pulposus and when surgical decompression of the cord or nerve roots is required, this can be accomplished better by laminectomy or laminectomy and foraminotomy. By this approach visualization can be adequate, and decompression can be certain. Secondly, as already mentioned in connection with Case 7 (Case 1, Table I) and as experienced in other instances where anterior cord decompression was performed for a destructive lesion of the vertebra, extradural bleeding may be profuse and difficult to control. With a laminectomy, bleeding vessels can be coagulated with electrocautery after controlling of the bleeding by compressing of the vessels against bone. With the anterior approach, compression and electrocautery cannot be employed.

Case C. This case has already been described in the section on disruption of the intervertebral disc.

Case 9. L. J. (Case 18, Table I), a skilled carpenter, thirty-eight years old, was first seen at University Hospital in May 1959 with complaints of pain in his neck and left arm. His difficulty began in June 1957 when he was struck by a cart while he was carrying two long sections of lumber on his right shoulder. The cart struck one of the pieces of lumber, producing a severe twist to the left of his neck. He experienced immediate pain in his neck, and within a week or so this extended to the upper outer aspect of his left arm. Because of persistence of these complaints, which were refractory to conservative measures, he was hospitalized in July 1957, and left hemilaminectomy of the sixth cervical vertebra was carried out with removal of a so-called protruded disc. His symptoms were promptly relieved in both his arm and neck, and he returned to full activity in one month.

He remained well until September 1958 when he slipped and fell, landing on his buttocks, with recurrence of similar symptoms. Again, he failed to respond to various forms of physical therapy and cervical traction.

On examination in May 1959, a distinct torticollis was present, with the head tilted to the left. Motions of the cervical spine were restricted in almost all directions, especially left lateral flexion and left rotation. Muscle spasm was palpable in the left posterior cervical muscles. The left biceps-tendon reflex was absent, and there was hypalgesia over the left sixth cervical dermatome. No muscle weakness was present. Jugular compression did not aggravate either the arm or neck pain.

Electromyography revealed evidence of irritation of the left sixth and seventh cervical nerve roots but did not indicate whether this was recent or of long standing.

Roentgenograms showed the hemilaminectomy with some decreased height of the intervertebral disc space between the fifth and sixth cervical vertebrae (Fig. 14-A). Recent myelograms that had been made at another hospital were normal.

Before cervical fusion was considered, the patient was immobilized in a Minerva plaster jacket

TABLE I

ANTERIOR FUSION OF THE CERVICAL SPINE

Case No.	Patient	Age (*Years*)	Date of Operation	Pathological Condition	Site of Fusion	Comments
1.	L.S.	21	Apr. 1952	Aggressive giant-cell tumor, with destruction at C4–5	C3–6	Fusion was successful; there was recurrence of the tumor, and the patient died two years later
2.	E.M.	54	Nov. 1955	Cervical spinal-cord meningioma was removed with cure twenty-five years previously; progressive quadriplegia due to spontaneous dislocation caused by instability secondary to previous laminectomy	C4–5	Fusion, with complete recovery from quadriplegia
3.	L.J.	17	Feb. 1956	Quadriplegia due to trauma at C4–5; laminectomy of C4–7 with resulting unstable spine	C4–5–6	Fusion; the patient is now rehabilitated and self-sufficient; he still has paralysis of the trunk and legs
4.	K.D.	46	June 1956	In Feb. 1953 a ruptured cervical intervertebral disc was removed by hemilaminectomy; in Sept. 1953 a second operation for disc removal was necessitated by a second injury; there was subsequent spontaneous dislocation with neck pain	C5–6–7	Fusion, with complete disappearance of neck pain
5.	J.C.	52	Feb. 1957	Fracture-dislocation, with dislocation of C5 on C6; previous reduction and application of Minerva jacket resulted in recurrence of deformity and rapidly developing quadriplegia	C5–6	Fusion through anterior approach was chosen because of extensive osteo-arthritic changes posteriorly
6.	D.R.	34	Jan. 1958	Hyperextension fracture-dislocation, with dislocation of C2 on C3, with bilateral acute temporomandibular dislocation and multiple fractures of the maxillae and mandible, with severe respiratory disturbance	C2–3–4	Fusion done with ease and with no respiratory difficulty during or after fusion
7.	B.N.	37	Feb. 1958	Fracture-dislocation, with dislocation of C6 on C7, with rapidly progressing quadriplegia; immediate laminectomy with dura left open	C5–6–7; T1	Fusion successful and done with ease; no respiratory difficulty during or after fusion; complete recovery from quadriplegia

8.	I.L.	37	June 1958	Destructive lesion of disc space between C3 and C4 in woman with known tuberculosis in many other areas	C3–4–5	Fusion obtained; patient had been on extensive antituberculosis therapy previously; no organisms were demonstrated in lesion by culture or guinea-pig study
9.	R.McG.	16	Aug. 1958	June 1958, fracture-dislocation, with dislocation of C5 on C6, with incomplete quadriplegia; immediate laminectomy accomplished with some difficulty; posterior fusion considered inadvisable at time of laminectomy	C4–5–6	Fusion gained readily; the patient has shown progressive improvement in hand function; there is little change in trunk and limbs
10.	J.C.	61	Oct. 1958	August 1958, fracture-dislocation, with dislocation of C6 on C7, with incomplete quadriplegia; no improvement after laminectomy with dura left open	C5-6–7	Fusion achieved; gradual return of function of the sixth and partial return of function of the seventh cervical nerve roots
11.	D.W.	24	Jan. 1959	One year previously, so-called whiplash injury with ascending quadriplegia relieved by laminectomy; neck pain and evidence of dislocation of C5 on C6 as shown by flexion-extension roentgenograms	C4–5–6	Fusion complete; no neurological abnormalities; patient entirely free from neck pain
12.	T.P.	42	Jan. 1959	Old (1950) fracture-dislocation, with spontaneous dislocation of C3 on C4 (1959); neck pain and questionable evidence of dynamic cord compression due to instability	C3–6	Fusion successful; marked improvement in cord-compression symptoms
13.	C.M.	22	Mar. 1959	Fracture-dislocation, with dislocation of C3 on C4, and fracture of the neural arch of C2; fractures of mandible, right frontal bone, and right leg; there were no neurological findings suggesting damage to cord	C2–3–4	Fusion obtained; anterior route chosen because of facial injuries and possibility that at a later date laminectomy might be required
14.	J.R.	45	Apr. 1959	Destructive lesion involving C5–6, with rapidly progressing quadriplegia	C4–5–6–7	Osteomyelitis due to *Pseudomonas aeruginosa;* recovery from quadriplegia has been slow with partial recovery in arms, but little change in lower extremities
15.	J.S.	14	May 1959	Extensive laminectomy (Oct. 1958) for the syndrome of anterior spinal-cord compression secondary to intradural extramedullary teratoma; there is instability of the spine secondary to extensive laminectomy as shown by roentgenogram	C4–5–6	The patient had shown complete neurological recovery from progressive quadriplegia after removal of a cervical teratoma; fusion was obtained with ease; the patient now appears to be normal

TABLE I - - *Continued*

Case No.	Patient	Age (*Years*)	Date of Operation	Pathological Condition	Site of Fusion	Comments
16.	J.P.	64	May 1959	Destructive lesion between C6–7 with rapidly progressing quadriplegia; laminectomy of C4–7 two weeks previously with no significant findings	C5–6–7	A large tuberculous abscess was found and decompressed; fusion now appears in progress; there is rapid improvement in neurological deficit, with complete return of function after operation
17.	R.S.	15	Aug. 1959	Fracture-dislocation with dislocation of C3 on C4, with fractures through the posterior neural arch of C2 in July 1959; the syndrome of central spinal-cord compression resolving	C2–3–4	Fusion progressing, there is slight weakness in the hands; there is complete return of function in both lower extremities; anterior fusion was selected because of the possible need for laminectomy in the future should any reversal in neurological status appear
18.	L.J.	38	Aug. 1959	In June 1957 there was twisting injury to the neck with brachialgia, which was relieved by laminectomy and removal of the disc in July 1957; there was a new injury in Sept. 1958, with recurrence of brachialgia	C5–6	The patient was relieved of neck pain while in a Minerva jacket; a disc was removed, and fusion performed anteriorly, with complete relief of the arm and neck pain
19.	J. W.	16	Oct. 1959	Instability of cervical spine secondary to previous extensive laminectomy (Aug. 1959) for cervical chordoma that had produced a Brown-Séquard syndrome; the latter was improving at the time of fusion	C4–5–6	Fusion progressing favorably; neurological improvement continues; patient still in neck brace
20.	D. M.	67	Oct. 1959	Lytic lesion of the body of C4, with cape hypalgesia; no other neurological findings; biopsy done as diagnostic procedure; bone-grafting	C3–4–5	Biopsy revealed lesion to be due to Hodgkin's disease; at two months, the postoperative area was treated with irradiation; when the patient was last seen the fusion was solid, and the patient was without complaints

for six weeks. This relieved his neck pain, but there was some persistence of his arm pain. Relief by immobilization served as an indication for fusion, which was performed through the anterior route. As much of the disc material was removed as possible. The nucleus was almost mucoid. Anterior fusion was performed between the fifth and sixth cervical vertebrae.

Almost immediately after surgery, the pain in the neck and arm subsided. Clinically and roentgenographically, the fusion appeared to become solid, and the patient returned to his regular activities without restriction (Fig. 14-B).

In this instance, a transverse instead of a longitudinal incision was used in the skin. We found that the transverse incision limits exposure in the longitudinal direction unless extensive mobilization of the skin is done. We prefer the longitudinal incision and have not found that it has resulted in scars that were unsatisfactory to the patient or surgeon.

SUMMARY AND CONCLUSIONS

This paper recounts chronologically the development and application of an original procedure for anterior fusion of the cervical spine. Twenty patients with various pathological lesions of the cervical spine were treated by this operation. No complications specifically related to this technique occurred. Fusion was achieved in all but two patients whose operations were performed less than six months prior to the time of writing. Table I summarizes our experience.

The value of posterior spine fusion as a means of re-establishing stability of the cervical spine has been proved. Since 1952 we have been exploring anterior fusion as an alternative to posterior spine fusion in instances where the posterior approach would be difficult, hazardous, or unsuited to the pathological condition at hand. With increasing experience the indications for anterior fusion have become broader, and the advantages of this procedure have become more clearly recognized.

In destructive lesions of the cervical vertebral bodies, anterior fusion is particularly useful. By this procedure it is possible to obtain biopsy material, to remove the lesion, to decompress the spine, and to restore stability.

In fracture-dislocations of the cervical spine anterior fusion is useful under the following circumstances:

1. After extensive laminectomies performed to decompress the spinal cord in acute disturbances, stability can be restored most effectively by anterior fusion;

2. In the presence of associated severe maxillofacial injuries that have caused or may cause impairment of respiratory function, anterior fusion can be performed with the patient supine, thus facilitating the maintenance of an adequate airway;

3. When pre-existing moderately severe or severe osteo-arthritic lipping indicates that disturbances in the function of the cord or nerve roots may occur in the future as the result of enlargement of the osteophytes and increasing foraminal encroachment, anterior fusion seems advantageous; once stability is established by anterior fusion the way is open for laminectomy, decompression, and foraminotomy should the need arise;

4. When the integrity of the intervertebral disc has been altered, and recurrence of the dislocation is a distinct possibility—a complication that has occurred in three of our cases—anterior fusion is a useful method of restoring stability.

Fusion of all fractures and fracture-dislocations that occur between the occiput and the first thoracic vertebra is not necessary; however, when fusion is indicated the relative ease of access to the involved vertebrae through the anterior approach, the lack of complication encountered in this series, and the rapidity with which fusion has occurred suggest that anterior fusion may be superior to the posterior procedure in many persons.

Spontaneous dislocations may occur early or many years after extensive laminectomy for the removal of a cord tumor, a ruptured intervertebral disc, or other lesion, especially if portions of the facets have been removed. These dislocations may cause disturbances in the function of the spinal cord or nerve roots,

which are apparently produced by a traction phenomenon secondary to the deformity of the spinal canal created by the dislocation. Posterior spine fusion under these circumstances is tedious and hazardous. The value of anterior fusion in restoring stability in these instances is clear.

In lesions of the intervertebral discs in the cervical spine we have limited use of the anterior approach to removal of the disc and fusion in two patients up to the time of writing.

Finally, we should like to emphasize that fusion of the cervical spine from an anterior approach has proved to be an excellent means of establishing stability. It is an excellent adjunct to posterior fusion. Both techniques are of extreme value, and the selection of which procedure to use must be based on the nature of the individual problem. Each patient reported here was treated according to his individual problems. As new problems arise, we believe that anterior fusion of the cervical spine will find wider applications.

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DISCUSSION

Dr. Robert A. Robinson, Baltimore, Maryland: In general, there are two major surgical routes to the cervical spine for fusion: the anterior route as used by the authors in the seven cases they report and the posterior mid-line route.

Three sites for fusion are available: (1) the posterior one in which the bone graft is applied subperiosteally to the laminae and the posterior spinous processes; (2) the posterior lateral one in which the bone grafts are applied to the facet-joint regions when the laminae and posterior spinous processes are absent; and (3) the anterior one in which the bone is either laid into a trough in the fronts of the cervical vertebrae or into the disc spaces. Sometimes, as in the authors' first case, where the bodies are eroded by tumor or infection, one must use the inlay, or in other cases combine the inlay and the intervertebral-body fusion techniques. Obviously, fusion occurs with the anterior-

(Continued on page 624)

Delayed Neurological Sequelae of a Retained Foreign Body (Lead Bullet) in the Intervertebral Disc Space

BY RUSSELL N. SHROYER, M.D., AND CHARLES H. FORTSON, M.D., DAYTON, AND C. B. THEODOTOU, M.D., COLUMBUS, OHIO

From the Orthopaedic Section, Veterans Administration Hospital, Dayton, and Ohio State University College of Medicine, Columbus

There have been a few papers in the past decade reporting the late onset of neurological symptoms due to foreign bodies embedded and retained in or about the spinal canal [2, 5]. This paper will present yet another case peculiar in regard to location of the foreign body and the reaction to it.

The illness began in 1948 when the patient was twenty-nine years old and was shot in the back, five centimeters to the left of the tip of the spinous process of the first lumbar vertebra with a .38-caliber pistol. This was followed by immediate paralysis of both legs, which lasted several hours. The patient was treated at a nearby hospital for three and one-half days. Penicillin was given, 50,000 units every three hours for two days and then 25,000 units every three hours for one and one-half days. He was then transferred to Brown General Hospital, Veterans Administration Center, Dayton, Ohio.

On admission the patient complained of severe pain in the lumbar area and tenderness of the left buttock. There was mild weakness of the left tibialis anterior, left quadriceps, and left hamstrings. There was no sensory loss. The ankle jerks were absent. The left knee jerk was absent. There was no disturbance of bladder or rectal function. Roentgenograms showed the bullet to have lodged in the disc space between the first and second lumbar vertebrae (Figs. 1 and 2).

The patient remained in the hospital for a month, receiving physical therapy. At the time of his discharge the pain and weakness had decreased, and the left knee jerk was present but weak.

The patient soon returned to work at a foundry. This work did not require any lifting, but he

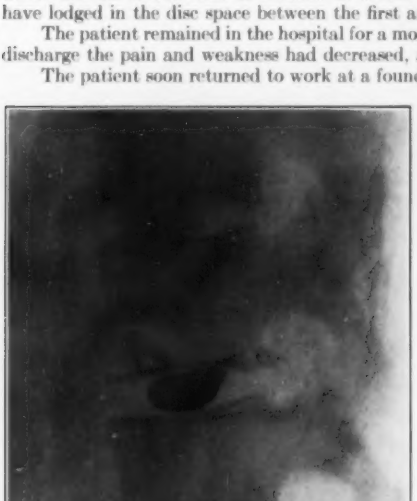

FIG. 1

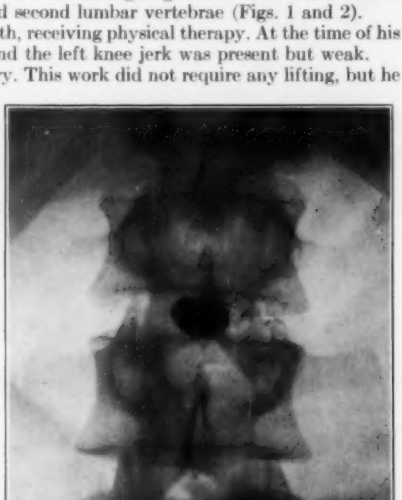

FIG. 2

Roentgenograms made in January 1948, several days after the injury, showing the bullet to have lodged in the disc space between the first and second lumbar vertebrae.

FIG. 3

FIG. 4

Roentgenograms made in August 1958, ten years after injury. A large radiopaque mass is seen extending posteriorly from the body of the first lumbar vertebra into the spinal canal.*

FIG. 5

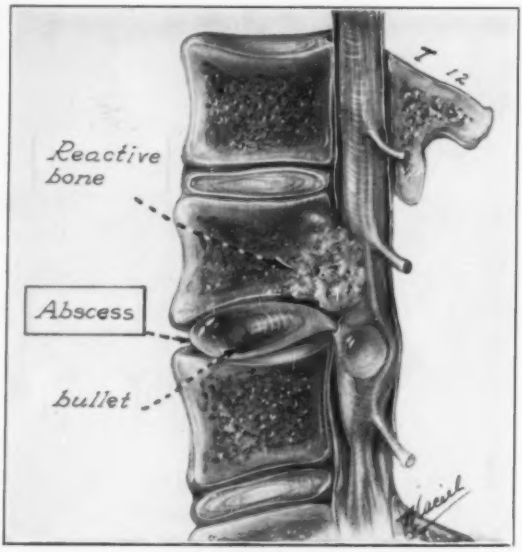

FIG. 6

Fig. 5: Myelogram made ten years after injury, showing a complete block.

Figs. 6: A schematic representation of the pathological condition seen at surgery. To be noted are: (1) the cyst or abscess cavity surrounding the bullet and projecting posteriorly into the dura; and (2) the radiopaque mass that projects posteriorly into the spinal canal, deforming the dura and contents.

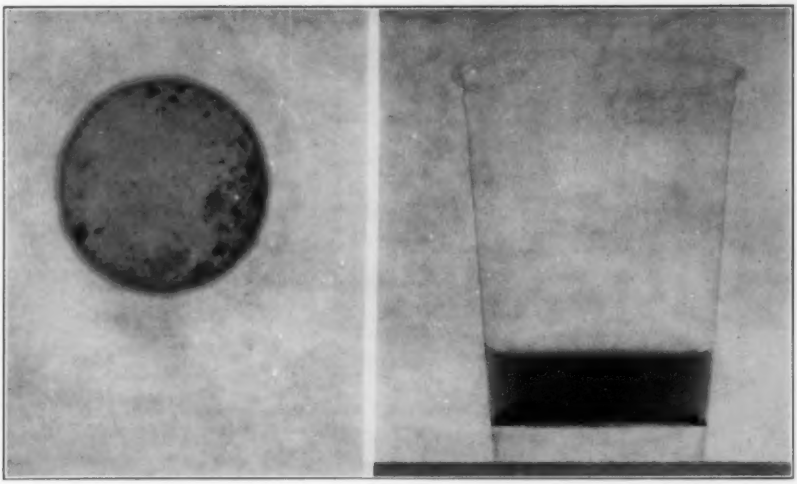

FIG. 7

Fig. 7: Roentgenogram of the calcareous portion of the specimen curetted from the vertebrae.
Fig. 8: Lateral view of the involved vertebrae six months after surgery.

FIG. 8

did perform jobs requiring much bending and stooping. He had no complaints and noted a gradual improvement of function. Subsequently, he was employed as a janitor and a drill-press operator and at one time was a member of the company baseball team. He did not detect any further difficulty until 1955, seven years after injury. At this time, while pitching for an amateur baseball team, he noted that he was unable to run rapidly and that his back was weak. He batted poorly from the right side and therefore began to bat from the left side. In 1956 the patient's back became slightly weaker, and during the 1957 season he began to suffer low-back pain on throwing a baseball, which caused him to stop playing.

Despite this he did continue to do hard labor on construction projects until one morning ten days before his return to the hospital in the fall of 1958 (ten years after injury). On arising on that morning he had severe pain in the lumbar area and in the left buttock. Examination elicited weakness of the right and left quadriceps that was more severe on the left. There was hypesthesia on the lateral aspect of the left calf. The right knee jerk was diminished, and the left was absent. Both ankle jerks were diminished. Straight-leg raising on the left was limited to 35 degrees, causing left-buttock and back pain. Roentgenograms again showed the bullet in the disc space between the first and second lumbar vertebrae, but the bullet now seemed smaller than ten years previously. In addition, a large amount of radiopaque material projected posteriorly into the spinal canal (Figs. 3 and 4). A spine tap revealed xanthochromic fluid with an initial pressure of 190 millimeters of water. The Queckenstedt procedure showed a complete block. After a few cubic centimeters of spinal fluid were removed the final pressure was 15 millimeters of water. The spinal-fluid protein was 1,440 milligrams, the globulin was four plus, and there were no cells. The myelogram showed a complete block at the level of the interspace between the first and second lumbar vertebrae (Fig. 5). At completion of the lumbar puncture and myelography the patient's neurological status was unchanged. A cystometrogram revealed a sensory atonic bladder.

Two days later a laminectomy was carried out from the twelfth thoracic to the second lumbar vertebrae. A hard mass, three centimeters by one centimeter, was palpated through the dura at the level of the first lumbar vertebral body. The mass was adherent anteriorly to the dura. On opening

of the dura, a cyst about one centimeter by one centimeter was found filling all available space within the dura and pressing the cauda equina on one side. The wall of the cyst had the appearance of a thin, pliable fibrous membrane. There were no adhesions or scarring of the arachnoid around the cyst, which was contiguous to the cauda equina. The cyst was separated easily from surrounding structures. There were no signs of inflammation about the area, but the cyst was filled with a yellow purulent fluid. The cyst communicated anteriorly through a small hole in the dura with a large cavity in the spinal canal at the same level. This cavity extended into the disc space between the first and second lumbar vertebrae (Fig. 6). The bullet lay free within this cavity in the disc space and was surrounded by the fluid contained in the cyst cavity. The bullet was smooth but somewhat flattened and was covered with a thin, grey, smooth crust that was easily scraped away with a knife, revealing soft glistening lead beneath. The radiopaque material, which on the roentgenogram occupied the inferior and posterior portion of the first lumbar vertebra and projected posteriorly into the spinal canal, was friable, grey, gritty, amorphous, and avascular; it was easily removed with the curette. As with the intradural portion of the cyst, there was no gross evidence of inflammation around this material. Most of the material, together with the bullet, was removed. The dura was closed with interrupted silk sutures without drains. Smear and culture of the fluid revealed no organisms. No acid-fast bacteria were found in the smear.

Two separate specimens were taken for microscopic study: (1) the soft tissue from the cyst wall around the bullet, and (2) the solid calcareous material that projected posteriorly from the vertebral body into the spinal canal. Microscopically, the soft-tissue specimen consisted of hyalinized fibrous tissue mixed with degenerated amorphous debris that stained slightly basophilic. There was no inflammatory reaction. There was a small amount of pigment that gave a positive stain for iron. Stains for calcium and lead salts were negative. Unfortunately, the specimen of the calcareous material was never subjected to microscopic study. A roentgenogram was made of the specimen (Fig. 7) but the specimen was then lost.

For the first few days after surgery there was some loss of muscle strength. Thereafter, the patient had gradual, progressive improvement. By the second month he no longer had pain, and there was no sensory loss. Some diffuse weakness of the entire right lower extremity persisted.

By the sixth postoperative month, he was still pain-free, with no sensory loss. At that time he still had a slight tendency to a drop-foot on the right, but muscle weakness was barely detectable and was confined to the right lower extremity. Slight weakness of the extensor digitorum communis, tibialis anterior, and tibialis posterior was observed. The deep-tendon reflexes of both lower extremities were absent. Roentgenograms now showed that the intervertebral space between the first and second lumbar vertebrae had narrowed moderately since surgery (Fig. 8).

DISCUSSION

It is interesting to observe the fate of the lead bullet that remained free in the intervertebral disc space for ten years while the patient engaged in strenuous labor and sports. It might have been anticipated that this "solid nucleus pulposus" would have been displaced either along the course of its entry through the annulus, or have been "herniated" through the relatively thin posterior annulus into the spinal canal. At the least, it might have been expected to have caused chronic severe low backache on strenuous activity. None of these things occurred.

The bullet was not unaffected. On comparison of the roentgenograms made soon after injury with those taken just before surgery, it appears that the bullet did decrease in size and became flattened, and that as this took place the amount of metallic debris in the contiguous tissue increased.

Retained foreign bodies of lead are generally considered to be inert. It appears, however, that when lead is exposed to a joint, either a physical or chemical process occurs, which may result in disintegration of the lead and a spread of lead particles throughout the joint. Key reported a case of foreign-body arthritis of the subtalar and talonavicular joints that developed about four years after a .22-caliber bullet became lodged in the head of the talus. The bullet had almost completely disintegrated, particles of lead were found in the cartilaginous surface of the head of the talus, and masses of amorphous material, presumably lead, were found throughout the extracellular tissues and in the deeper subsynovial tissues. Leonard, Breck, and Kosicki also reported a few cases in which lead bullets that became lodged in joints subsequently broke up into small particles that were dispersed throughout the joint capsule.

Key also reported a case in which a rifle bullet, apparently lead, had lodged in the intervertebral disc space between the first and second lumbar vertebrae.

The patient was asymptomatic when seen twelve years later, and roentgenograms showed that the disc space had disappeared, with fusion of the bodies of the second and third lumbar vertebrae. In this instance, in which fusion had taken place, the amount of osseous or calcific reaction to the foreign body was mild. The bullet itself apparently had not changed in size or shape during the twelve-year period.

In the case presented in this paper the lead bullet did appear to become smaller and flattened. A mass of radiopaque, gritty, amorphous, avascular material formed in the postero-inferior portion of the first lumbar vertebra and projected posteriorly into the spinal canal. Unfortunately, microscopic and chemical analysis of this material was not performed. Roentgenographic examination of the specimen covered with water in a glass showed an aggregation of semiopaque particles that was assumed to be amorphous calcium or necrotic cancellous bone (Fig. 7). A cyst containing sterile yellow purulent material surrounded the bullet and projected posteriorly into the dura. Microscopic section of this cyst wall did not show evidence of inflammation or the presence of lead.

Whether the observed decrease in the size of the bullet was caused by the mechanical grinding effect of the adjacent vertebrae, or by the dissolving of the lead by the tissue fluids, or by a combination of these mechanical and chemical processes is not determined. In any event, a low-grade sterile inflammatory process eventually developed, which resulted in the reaction of the soft tissue and bone. It has been well demonstrated that even "inert" metals evoke a reaction in the subcutaneous tissue when implanted as a very fine powder suspension, and it seems possible that the unusual reaction to lead in this instance was due to lead "dust" dispersed into the contiguous tissues [1].

Finally, it is our belief that this case corroborates the conclusion of Schneider and Kahn ". . . that early careful surgical removal of foreign bodies from the spinal canal with associated preoperative and postoperative antibiotic therapy is desirable in order to prevent chronic infection or increased bone overgrowth with its attendant spinal-cord compression". To this we would add cauda equina compression.

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The Deformity of the Medial Tibial Condyle in Nineteen Cases of Gonadal Dysgenesis

BY JERZY KOSOWICZ, M.D., POZNAŃ, POLAND

From the Second Department of Internal Medicine of the Medical Academy, Poznań*

Gonadal dysgenesis, the result of faulty development of the gonads in fetal life, occurs only in persons of feminine appearance. It is encountered in three different types of persons: (1) in eunuchoids; (2) in patients with numerous congenital anomalies, including short stature—the so-called Turner's syndrome; and (3) in normal-appearing females. Previous studies showed that skeletal changes in Turner's syndrome are very frequent and are of value in the diagnosis of this disorder [3]. One of the most common of these changes is an enlargement and de-

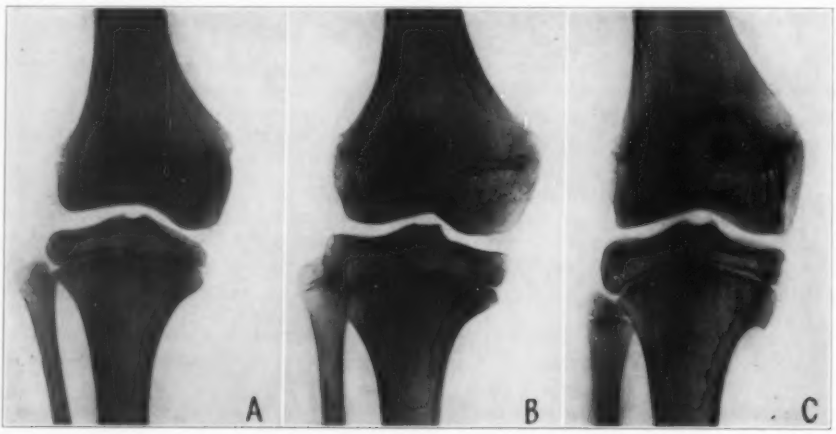

FIG. 1

The stage of the disease in childhood.

A. The tibial metaphysis shows enlargement medially in the form of a mushroom with depression of the plateau on the medial side. There is some flattening of the lateral femoral condyle.

B. Another instance, demonstrating projection of the metaphysis beyond the secondary center of ossification of the epiphysis. The fibular head projects higher than normal. The lateral portion of the tibial epiphysis shows an exceptional pointed contour on its inferior lateral border.

C. Another instance, demonstrating the blunt enlargement of the medial tibial metaphysis.

formity of the medial tibial condyle similar to that found in Blount's disease. I have described this associated finding in twelve of eighteen investigated cases of Turner's syndrome [4]. A larger number of patients and a longer period of observation were required to determine more exactly how the changes in the medial tibial condyle evolved, what was their course, and what were the late sequelae.

Twenty-eight patients with gonadal dysgenesis were examined, of whom four were eunuchoid, and twenty-four of whom had Turner's syndrome. None of the eunuchoid patients showed the changes in the medial tibial condyle to be described, and they will not be considered further. The ages of the other patients ranged from six to forty-two years. The diagnosis of gonadal dysgenesis was made on clinical grounds, confirmed by at least one of the following findings: increased output of gonadotropins in the urine, male chromatin pattern in the oral-mucosa cells, or typical streak gonads demonstrated by laparotomy.

* Chief of Medical Department, Jan Roguski, M.D.

FIG. 2

Three cases illustrating the stage of deformity in adolescence. All roentgenograms show pointed projections of the medial portion of the tibial epiphysis. The medial femoral condyle is lower than normal because of a depression of the tibial plateau. The fibular head is higher than normal.

All but two of the patients were asymptomatic with reference to their knees, and those two had pain, possibly due to osteoporosis, that subsided after treatment with estrogens.

The lower extremities of the patients were thick and stubby. In only a few was the fibular head palpable closer to the knee joint than normal. In two patients there was slight genu varum. The range of motion of all of the knee joints was normal.

Roentgenographically, a deformity of the medial tibial condyle was present in nineteen patients. In the remaining five patients no such change was found. Three degrees of change may be differentiated: that observed in childhood; that in adolescence; and that in adulthood. In the first stage only the metaphysis is affected; in the second, the epiphysis is also involved; and in the last, the final deformity occurs as a result of the fusion of the epiphysis and metaphysis.

The first stage (in childhood) (Fig. 1, *A*, *B*, and *C*) was found in patients aged six to fourteen years. The medial portion of the metaphysis either showed

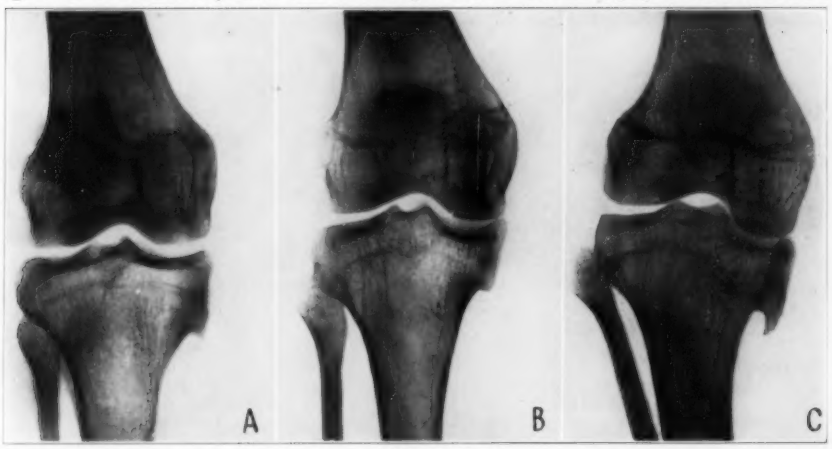

FIG. 3

The stage in adulthood, showing a blunt projection of the medial tibial condyle in *A* and *B* and a beaked projection in *C*. The medial femoral condyle is depressed, and the fibular head elevated.

FIG. 4-A FIG. 4-B

Figs. 4-A through 4-D: Development of deformity of the medial tibial condyle in the same patient at the age of thirteen (Fig. 4-A); fifteen (Fig. 4-B); sixteen (Fig. 4-C); and twenty-two years (Fig. 4-D).

Fig. 4-A: The blunt enlargement of the tibial metaphysis is the sole deformity.

Fig. 4-B: An accessory ossification center of the tibial epiphysis is evident medially and is part of the pointed overhanging projection of the epiphysis. The other changes of the femoral condyles and the metaphysis are also evident.

protrusion medially or was enlarged in the form of a mushroom. In some cases the medial tibial condyle was slightly depressed. Coordinate changes in the femoral condyles resulted in a flattening of the lateral femoral condyle and a rounded contour and more inferior position of the medial femoral condyle. The tibial epiphyseal plate was altered from its straight and slightly bowed normal contour

FIG. 4-C FIG. 4-D

Fig. 4-C: The separate ossification center has fused to the main ossification center of the epiphysis.

Fig. 4-D: The stage in adulthood with a blunt projection of the tibial condyle, flattened lateral femoral condyle, depressed medial femoral condyle, and high fibular head.

and became curved inferiorly so that its medial border was depressed. The roentgenographic diagnoses in the early stages were difficult to make unless alterations in the epiphysis were present in addition to those in the metaphysis.

In the second stage, in patients twelve to sixteen years of age, the changes in the metaphysis were accompanied by closely related alterations in shape of the epiphysis (Fig. 2, *A*, *B*, and *C*). The medial portion of the epiphysis spread out medially, covering the enlarged metaphysis either partially or completely and, in the latter instance, often continued to project inferiorly. Rarely, this inferior projection of the epiphysis constituted an additional ossification center that persisted only for a short time before it fused with the remaining portion of the epiphysis. These changes in the metaphysis and epiphysis usually were bilateral and symmetrical. The roentgenographic bone texture and density of the medial tibial condyle did not differ from those of other parts of the tibia. In about half of the patients some atrophy was evident, not localized to the area under discussion. In some instances the fibular heads were situated higher than normal.

In the third stage, found in adulthood, fusion was evident between the epiphysis and metaphysis. In untreated patients this fusion was delayed, but with treatment by sex hormones, fusion occurred at about the normal age. During this stage the irregular curvature of the fused epiphyseal line indicated the antecedent stages just described. The medial projection of the tibial condyle often assumed a beaklike shape that varied in size and resembled an exostosis. This projection was blunt in most instances—that is, when the projection of the epiphysis had completely covered the deformed metaphysis. It was beaklike when only partial covering of the metaphysis had occurred. The other features just described—depression of the medial tibial plateau, flattening of the lateral femoral condyle, superior location of the fibular head, and bone atrophy—also were evident. Infrequently, the lateral tibial condyle also was slightly enlarged, but to a lesser degree than the medial.

DISCUSSION

The frequent occurrence of a deformity of the medial tibial condyle in gonadal dysgenesis, which has just been described, and also as reported by Overzier, is part of a more generalized process affecting the epiphyseal plates in other bones as well. In the distal radial metaphysis, for instance, the epiphyseal plate may be irregularly arched with corresponding changes in the epiphysis, which then becomes narrowed centrally to compensate for the abnormal shape of the cartilaginous epiphyseal plate. There infrequently develops premature fusion of the epiphysis to the metaphysis laterally, resulting in Madelung's deformity. The distal part of the ulna may also show obliquity. In about half of the patients, the epiphyseal plates of the vertebral bodies are irregular. The irregularity is partially compensated for by reciprocal irregularities of the secondary centers of ossification of the bodies of the vertebrae, but usually some narrowing of the intervertebral spaces is evident. In the femoral neck, the epiphyseal plate for the femoral capital epiphysis may be uneven and wavy, with compensatory adaptive changes in the femoral head.

Of all the deformities mentioned, that of the medial tibial condyle is the most common. Only in that area is there a permanent disturbance of contour in the form of a blunt projection or beaklike exostosis. Why the generalized irregularity of the epiphyseal plates is most frequently demonstrable in the medial tibial condyle is difficult to explain. Perhaps, weight-bearing and the large size of this epiphyseal plate are responsible.

In differential diagnosis, two disorders should be considered: diaphyseal aclasis and Blount's disease (tibia vara). Diaphyseal aclasis affects many bones, especially the femora, and exostoses never develop in the epiphyses[2]. In contrast, in gonadal dysgenesis the epiphyses take part in the formation of the deformity; although exostoses occur in the tibiae, no exostoses in the femora are seen. In

Blount's disease, the roentgenographic changes are quite similar but frequently are unilateral, whereas in gonadal dysgenesis the changes are always bilateral and symmetrical. Genu varum is much more commonly seen in Blount's disease.

SUMMARY

In nineteen patients with Turner's syndrome (gonadal dysgenesis associated with short stature and congenital anomalies), bilateral symmetrical deformity of the medial tibial condyle was found. In five other patients and in four eunuchoids, such changes were not observed. Three stages of the deformity were distinguished. In the stage that occurs in childhood there was an enlargementof the tibial metaphysis. In the stage in adolescence the adjacent portion of the epiphysis became pointed and resembled an exostosis. In the stage in adulthood a blunt projection or beaklike exostosis of the medial tibial condyle remained. The deformity of the medial tibial condyle is part of a generalized process involving the epiphyseal plates of bones in gonadal dysgenesis.

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The Orthopaedist and Apical Lung Carcinoma*

BY PAUL E. DEE, M.D., R. GLENN SMITH, M.D., MILES J. GULLICKSON, M.D., AND CHARLES S. BALLINGER, M.D., ROCKFORD, ILLINOIS

The unique symptom complex of tumors of the thoracic inlet (superior sulcus tumors) was described by Pancoast in 1932. The most striking characteristics of the Pancoast syndrome are involvement of the brachial plexus and the cervicothoracic sympathetic trunk and involvement of the upper ribs and adjacent transverse processes. Browder and DeVeer, in 1935, presented cases which indicated that this syndrome is not caused by a single pathological entity, but that in this area tumors and diseases of several types may result in similar symptoms. It was soon recognized that a large proportion of tumors producing the Pancoast syndrome were apical lung carcinomata [5].

Herbut and Watson recorded a series collected from the medical literature of 134 tumors of the thoracic inlet and added seventeen of their own. Of the 151 patients, 100 had carcinoma of the lung. The following quotation from Herbut and Watson's paper is singularly appropriate as an expression of the seemingly innocuous nature of the symptoms that precede the full development of the Pancoast syndrome: "Frequently, however, the patient has consulted a physician when pain in the shoulder and arm first developed, and because an obvious cause could not be found the condition has been diagnosed for months as neuritis. . . . It appears, therefore, that the cause of any persistent neuritis in the shoulder or the arm should be thoroughly investigated, even by surgical exploration if necessary."

A number of similar experiences of our own have stimulated our interest in the frequency of pain in shoulder, arm, or neck as an early symptom of pulmonary carcinoma. The increasing prevalence of lung carcinoma has resulted in its displacing carcinoma of the stomach as the most common carcinoma in the male American. Of 649 carcinomata classified by Simons as to lobar involvement, 348 (54 per cent) were of the upper lobes. The incidence of true apical carcinoma of the lung is not known. However, any tumor, benign or malignant, as well as some inflammatory processes in the upper lung or mediastinum, can produce all or a portion of the symptoms of apical lung carcinoma.

Cough, hemoptysis, dyspnea, chest pain, and loss of weight are considered the most frequent symptoms of carcinoma of the lung. In the apical lung carcinoma these may often be absent. The typical features of the Pancoast syndrome—severe shoulder pain, Horner's syndrome, recurrent laryngeal-nerve paralysis, wasting of the small hand muscles, and erosion of the ribs or vertebrae—are the late manifestations of apical lung carcinoma. The apex of the thoracic cavity is small, and invasion of extrapulmonary tissues by the tumor occurs early. The predominant early symptom is vague shoulder discomfort. This is owing to involvement of the parietal pleura or peripheral branches of the intercostal nerves and brachial plexus. The persistence of this discomfort in the shoulder, neck, or arm causes the patient to consult a physician. Treatment, such as diathermy and massage or cortisone, is often instituted. If specific incidental disease, such as cervical arthritis or calcification in the subdeltoid bursa, is found therapy is directed toward that condition.

This sequence of events has occurred with several of the patients we saw during the past year. The interval between the first medical consultation and recognition of the true nature of the disease for our patients has varied from sev-

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 25, 1960.

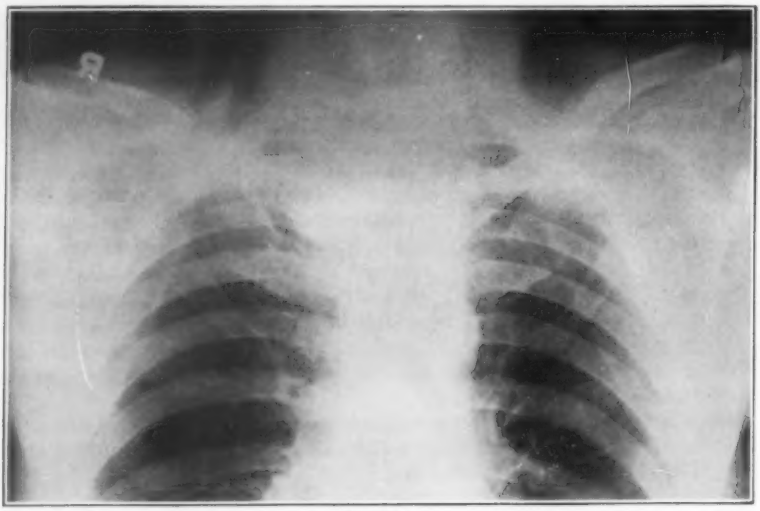

FIG. 1-A

Case 1. A slight opacity of the right apex, as compared with the left apex, indicates the presence of this apical carcinoma.

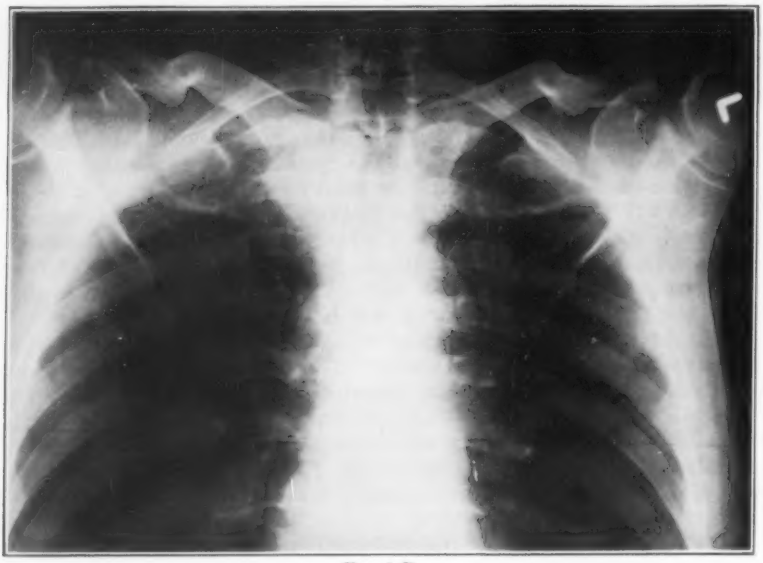

FIG. 1-B

A lordotic roentgenogram of the chest more clearly demonstrates the carcinoma in the apex of the right lung.

eral weeks to several months. Usually, the appearance of one of the late symptoms just described first aroused suspicion of the more serious disease. In a recent series reported by Haas and associates the average time interval between the appearance of the first symptom and the time when the correct diagnosis was established was 10.1 months, varying from one-half to thirty-two months.

Unfortunately, a roentgenogram of the chest will not always result in a correct diagnosis. In a high percentage of cases the correct diagnosis is not made from the first roentgenograms of the chest. (Only in retrospect is this lesion recog-

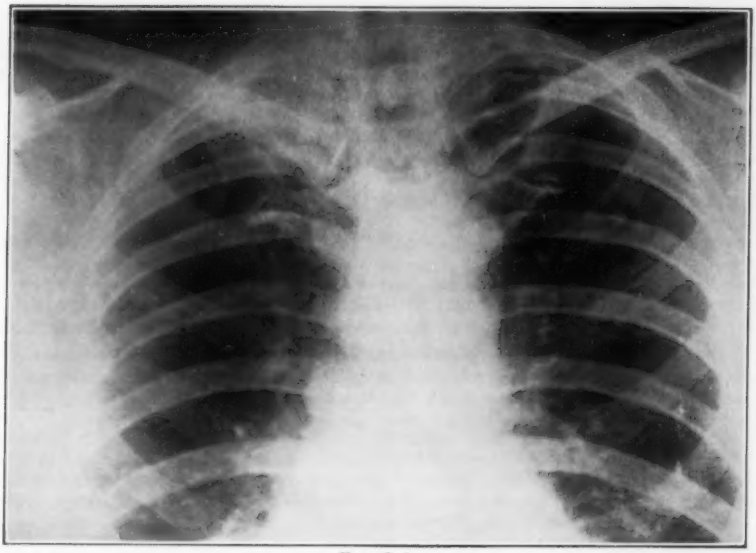

FIG. 2-A

Case 2. A review of this roentgenogram, made in April 1955, originally reported as essentially negative, called attention to the probable association of the opacity in the right apex with pain in the shoulder.

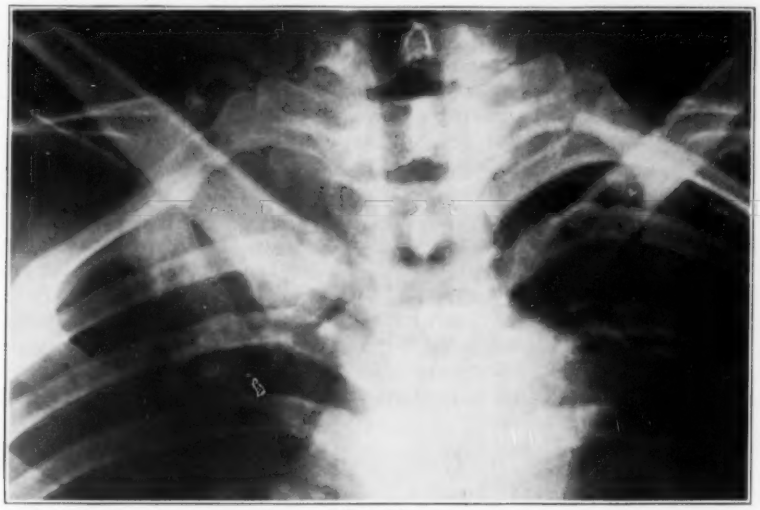

FIG. 2-B

This roentgenogram, made in July 1955, shows increased density of the right apex with destruction of the posterior portion of the right second rib.

nized as such.) The extrapulmonary tissues in this small space are involved early, with the result that the extent of pulmonary lesion may be quite small when the clinical symptoms first appear. Thickening of the pleura at the apex or insignificant pulmonary infiltration may be overlooked or mistaken for a quiescent acid-fast infection. It is evident that apical lung carcinoma offers unusual difficulties in early diagnosis because of the paucity of early clinical symptoms and roentgenographic findings that clearly indicate pulmonary carcinoma. The following brief case histories illustrate this point:

CASE 1. R. L., a man, fifty-nine years old, received some minor injuries in an automobile accident on May 15, 1957. His principal complaint was pain in the right shoulder and the right side of the neck. This pain became steadily worse, and, in the first part of June 1957 he consulted a physician and was hospitalized. Roentgenograms of the shoulder, cervical spine, and chest were made and a diagnosis of cervical arthritis was established. The patient was treated with diathermy and cervical traction. After ten days, because he had obtained no relief, he left the hospital at his own request and was thereafter treated as an out-patient. The first week in August he consulted his present family physician. Horner's syndrome on the right side was noted at that time. On August 12, 1957, he was readmitted to the hospital. A second roentgenogram of the chest (Fig. 1-A) was made and reported as negative. Laryngoscopy now demonstrated a paralysis of the right recurrent laryngeal nerve. Physical examination disclosed a slight fullness in the right supraclavicular area and some tenderness. The roentgenograms of the chest were reviewed and lordotic views (Fig. 1-B) were obtained. The diagnosis of apical lung tumor with some displacement of the esophagus and the trachea was then made and on August 21, 1957, a resection of the upper lobe of the right lung was performed. The histological diagnosis was adenocarcinoma of the right upper lobe.

CASE 2. H.D.C., a white man, sixty-seven years old, in February 1955 complained of a chronic non-productive cough, a fifteen-pound weight loss, and numbness on the posterior aspect of the right arm, the anterior surface of the right forearm, and the volar surface of the index finger. In April 1955 pain in the right shoulder was said to be the most troublesome symptom. The patient was treated intensively by diathermy, cortisone, and other medication. Roentgenograms of the chest made in April (Fig. 2-A), June, and July of 1955 were reported as negative. Later in July 1955 a destructive lesion in the posterior portion of the right second rib was reported. Six weeks later the patient was seen by a thoracic surgeon and a diagnosis of advanced apical carcinoma of the lung was made (Fig. 2-B). Since no evidence of distant metastasis could be discovered, a surgical procedure, consisting in block resection of the right upper lobe and the upper four ribs, was done.

The incidence of carcinoma of the lung is increasing at an alarming rate. It may be anticipated confidently that every orthopaedist will see patients with apical lung cancer whose initial complaint will be pain in the shoulder, arm, or neck. Patients at the age when cancer is common may well have associated benign bone or joint lesions which will divert attention from the true cause of the pain. The significant incidence of patients with carcinoma of the apex of the lung in our own experience leads us to recommend that in every series of roentgenograms made because of shoulder pain there should be a postero-anterior view on a film large enough to include the upper portion of the chest and mediastinum on the same side.

Although apical lung cancer has a relatively unfavorable prognosis, shoulder, neck, or arm pain can also be the initial symptom of more favorably located lung cancer and of benign tumors of the mediastinum, the upper part of the chest, or lower part of the neck. A recent excellent summary of the problem of the painful shoulder [2] accords but scant mention to tumors of the superior pulmonary sulcus and none at all to pulmonary carcinoma. Improvement in the rate of cure of apical pulmonary carcinoma can result from earlier diagnosis by thoracic surgeons and roentgenologists and from a higher index of suspicion among general practitioners and orthopaedists, who are often the first to see patients with pain in the shoulder, arm, or neck.

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(Discussion is on page 636)

Simple Bone Cysts

Studies of Cyst Fluid in Six Cases with a Theory of Pathogenesis

BY JONATHAN COHEN, M.D., BOSTON, MASSACHUSETTS

From the Departments of Orthopaedic Surgery and Pathology and Division of Laboratories and Research, Children's Medical Center, Boston

Simple or solitary cysts of bone have been recognized as a pathological entity for at least four decades [2]. The prime requisite to the diagnosis is that the lesion be a large cavity within bone which is filled with fluid. Other characteristics, such as the presence of a distinctive lining membrane, the usual localization in the metaphysis, and the onset in childhood, are well known and contribute to definitive diagnosis. The present study of the characteristics of the cyst fluid in six cases was undertaken in the hope that it would shed some light on the pathogenesis and character of the lesion beyond that derived from previous intensive pathological, roentgenographic, and clinical studies.

The descriptive data on the clinical manifestations and course, the roentgenographic appearance, and the histological variations of simple cysts of bone have not been added to appreciably during recent years. The older descriptions, exemplified by that of Bloodgood, usually included other lesions, such as aneurysmal bone cysts and osteitis fibrosa generalisata. Newer clinical investigations and textbook accounts have established these as separate entities and thereby delimited the classification of simple cysts more sharply. From the standpoint of pathogenesis and etiology, however, no new data have appeared. Jaffe's textbook on bone tumors [7] includes a chapter on the simple cyst, although admittedly, as he notes, it "is not a tumor in the strict sense". Several etiological speculations have been proposed, but none fits all the facts or has gained wide acceptance.

The only information which could be found in the literature concerning cyst fluid from bone cysts is descriptive. In most cases the fluid is described as clear or slightly cloudy, viscous, or of low viscosity, and yellow, yellow-green, or straw colored. Occasionally, accounts state that the fluid coagulates on standing. In some instances, but not in all, when fractures had occurred through the cyst, the fluid obtained resembled blood in appearance and, on occasion, contained clots. Serosanguineous fluid has also been described. For proper evaluation of the present study it is necessary to assume that cysts may vary in the type of fluid they contain either because of the nature and rate of the formative process or because of trauma. It may be impossible to prove that fluid with an appreciable admixture of blood has come from a cyst which has not suffered significant trauma either through fracture or by the surgical entry into the cavity. In the literature the procedure by which any single type of cyst fluid was obtained is rarely given in sufficient detail to evaluate the possible influence of trauma, particularly when the fluid resembled blood or was serosanguineous.

For each of the six cases studied in this report, a detailed description of the fluid and its manner of procurement will be presented in addition to a brief case report. The series represents consecutive cases with histological verification of diagnosis, in which adequate quantities of fluid and venous blood were obtained for the examinations listed. Standard laboratory methods were used for all the analyses.

Case 1. W. L., a boy, was first hospitalized when he was eight years old with the history of pain in his right humerus of four years' duration. At the age of six he fell and fractured the humerus; a cystic lesion at the fracture site was demonstrated by roentgenogram. The fracture united under

conservative treatment, but the cyst increased in size. Once the fracture had healed, the only positive physical finding was slight fullness in the upper arm. Routine laboratory tests were within normal limits. Repeated roentgenographic examinations showed that the cyst occupied the upper six centimeters of the metaphysis and diaphysis of the humerus. The humeral shaft was expanded and irregular in outline. During the year before hospitalization the cyst showed slight enlargement from six to 7.5 centimeters in length. A narrow zone of ossification separated the cyst from the epiphyseal plate. No additional bone had been laid down proximal to the cyst in that time. Because of the enlargement of the cyst and the continued discomfort, surgical removal of the lining of the cyst and packing by bone-bank bone grafts of irradiated ribs was performed. No description of the cyst fluid at that operation was recorded. The pathological specimen was a tough membrane several millimeters thick which on histological examination showed the loose fibrous tissue and hemosiderin accumulation typical of simple cysts, together with a lining layer of elongated fibroblasts and many thin-walled endothelial-lined sinusoids underneath. Five months postoperatively, a recurrence of the cyst was evident. One year following the operation, two centimeters of normal bone had been laid down between the epiphyseal plate and the recurrent cyst while the diameter of the humerus in the cystic area had increased from 2.2 centimeters to 2.8 centimeters. A second surgical procedure was done. After the periosteum was exposed and reflected, a hole was drilled into the cyst cavity and the contents were evacuated. Aspiration was necessary to remove the fluid. It was straw colored and coagulated on standing. Sixteen cubic centimeters of fluid were obtained. No analysis of the fluid was done. The cavity was then unroofed, the cyst lining was curetted, and irradiated bone-bank bone rib grafts were again packed into the cyst cavity. The pathological examination of the lining revealed a tissue similar to that previously described with no inflammatory reaction. A few giant cells were seen in the membrane. Six months later a recurrence of the cyst was noted. During the next year it showed a slight increase in diameter and radiolucency but no further increase in length. The patient continued to have minor aching in the arm until a new fracture occurred when he was fifteen years old—five years after the second surgical procedure. This fracture healed with slight angulation. Apparently, repair bone divided the cyst into compartments so that a number of separate areas of radiolucency now were evident by roentgenogram. A third operative procedure was performed during which two separate cavities were found. Each cavity was aspirated carefully with a 16-gauge needle through the thin cortical wall. In both cavities fluid resembling blood was encountered. This fluid would not flow out through the needle and had to be aspirated. The two fluids were pooled for analysis. After aspiration each cavity was unroofed. In each, a lining grossly similar to that previously described was encountered; a bone partition separated the two cysts. The histological structure of the lining was as just described. The cysts were filled with bone chips from the patient's ilium. The last operation was too recent for follow-up and evaluation of the end results.

Case 2. A girl, T. E., was five years old when she fractured the proximal end of the right humerus. Roentgenograms showed a bone cyst in the upper end of the humerus. The fracture was displaced. The cyst involved the proximal third of the diaphysis and extended up to the epiphyseal plate. During the next year and one-half there were six episodes of trauma to this area with probable fractures. All of these were treated conservatively by a sling, and healing of the fractures occurred. During the next four years, the cystic process showed signs of healing. The radiolucency diminished in intensity and in extent, and the upper limit of the cyst became separated from the epiphyseal plate by a zone of normal bone. An eighth fracture occurred in the cystic area when the patient was ten years old. This healed without incident, and two months later the patient underwent surgical treatment of the cyst. Physical examination then showed some atrophy of the shoulder musculature and slight limitation of motion of the shoulder. There was palpable thickening of the upper end of the humerus with no other abnormal findings. There was 2.3 centimeters of shortening of the right arm. Laboratory examinations were within normal limits. A skeletal survey was negative. The periosteum was exposed and a trocar was inserted into the cystic area. Twenty cubic centimeters of fluid resembling blood was aspirated. This fluid did not enter the syringe spontaneously nor did it pulsate. It coagulated in the tube several minutes after removal. The membrane of the cyst was curetted and bone-bank irradiated-rib grafts were inserted into the cavity. Bleeding into the cavity was brisk until all of the membrane had been removed. Histological examination of the cyst lining showed loose fibrous tissue, mononuclear cells, giant cells, a fairly distinct lining layer of fibroblasts, and small collections of hemosiderin. Many thin-walled endothelial-lined vessels were found in the membrane. This case is too recent for follow-up evaluation.

Case 3. A boy, L. W., aged five was first seen because of pain in his left hip after a fall. Roentgenograms demonstrated a cystic lesion of the upper end of the shaft of the femur adjoining the epiphyseal cartilaginous plate of the greater trochanter. At another hospital this cyst was evacuated and packed with bone chips. No record of the appearance of the cyst fluid at that time is available. The cyst recurred after several months and thereafter the patient had repeated roentgenographic examinations, all of which showed a radiolucency measuring 4.5 by 2.5 centimeters with a sclerotic border, a thin cortex, and no expansion of the diaphysis. The cyst grew slightly but normal bone began to be laid down between it and the epiphyseal cartilage. When the boy was nine years old, a second operation was performed. The surgical exposure was developed down to the cortex and a small drill hole was made into the cavity. Yellowish clear fluid emerged seemingly

under very little pressure, and aspiration yielded seventeen cubic centimeters of this fluid. The cyst was then unroofed and its wall was curetted prior to filling the cavity with irradiated bone-bank rib grafts. Histological examination of the tissue showed fragments of fibrous connective tissue two to five millimeters in thickness with a definite fibroblastic cellular lining and thin-walled vessels. Aggregates of mononuclear cells and cholesterol clefts were identified in the tissue fragments, and giant cells were also seen. Follow-up one year later showed good incorporation of the grafts and obliteration of the cyst.

CASE 4. L. F., an eight-year-old boy, was well until he fell and fractured his right humerus. A cyst which bordered on the epiphyseal plate was noted on the roentgenogram in the area of fracture. A roentgenographic skeletal survey revealed no other lesions. Conservative treatment resulted in the healing of the fracture but the cyst became a little larger expanding the humerus slightly. During the next year and one-half the interval between the epiphysis and the cyst showed an accumulation of normal bone. When the boy was nine years and four months of age physical examination showed minimum enlargement of the upper end of the right humerus. The laboratory findings were within normal limits. The roentgenogram showed a cystic lesion in the humeral metaphysis which measured approximately six centimeters in length and 3.3 centimeters in width. It was irregular in shape and appeared to be trabeculated. The cortex was eroded but no evidence of recent fracture was present. The lesion was approached surgically and before the periosteum was incised a No. 19 needle was easily inserted into the cyst. Fluid did not flow from this needle spontaneously but aspiration easily yielded ten cubic centimeters of fluid. The first sample of fluid, which amounted to seven cubic centimeters, was yellow and was similar to plasma in viscosity. The subsequent sample of three cubic centimeters was admixed with blood. Further aspiration of the cavity yielded fluid resembling venous blood. All of the samples mentioned coagulated on standing a few minutes. The first sample was that analyzed chemically. After the aspiration the cyst cavity was opened, a thin lining membrane removed with a curette and the cavity filled with irradiated bone-bank rib grafts. Pathological examination of the membrane showed mostly fibrous tissue with a fibroblastic lining, some giant cells, and evidence of recent and old hemorrhage. Thin-walled sinusoids also were demonstrable. The case is too recent for adequate follow-up data.

CASE 5. C. J. was first seen at the age of nine because of a fracture of the right radius. Roentgenographically the fracture traversed a bone cyst in the distal quarter of the right radius which expanded the cortex and extended to the distal epiphyseal cartilage. Slight trabeculation was evident. The patient was treated conservatively for four and a half years during which time no further fracture occurred, and a small layer of bone appeared between the epiphysis and the cyst wall. When the boy was twelve years old it was decided to treat the lesion surgically since there had been some expansion of the lesion. Physical examination prior to surgery showed only slight widening of the radius in the involved region caused by a non-tender bone mass. Some limitation of motion was evident as well as angulation of the radius. Skeletal survey was negative and laboratory examinations were within normal limits. The lesion was approached surgically and when the periosteum was elevated, inadvertently a thin flake of cortex was depressed but no fluid was seen to emerge. Removal of a small section of cortex was then done, exposing a membrane. Through this membrane fifteen cubic centimeters of amber-colored fluid was aspirated through a No. 18 needle. This fluid was not under tension and would not emerge into the syringe spontaneously. The fluid, on standing, coagulated. The window of the cyst was then enlarged and the membrane curetted thoroughly. The membrane was very thin and extended to the periosteum in the ulnar portion of the radius, no cortex being present in this area. The cyst wall was curetted thoroughly and irradiated bone-bank rib grafts were inserted to fill the cavity. Histological examination of the cyst wall showed a very thin layer of dense connective tissue lined by elongated fibroblastic cells. Large endothelial-lined channels were prominent. A few giant cells were seen. The histological diagnosis was simple bone cyst.

CASE 6. D. G., a boy aged four and a half, began to limp; roentgenograms showed a cystic lesion in the left femur. He was admitted to the hospital one month later for surgical exploration. Physical examination revealed slight atrophy of the left thigh and about one-half inch increased length of the left lower extremity. The laboratory findings were within normal limits. Roentgenograms of the left femur showed an irregular rounded lesion three centimeters in diameter in the intertrochanteric area. On one view, slight expansion of the medial portion of the neck of the femur by the lesion was evident, as was a thin fracture line. The margins of the lesion were smooth and not sclerotic and did not touch the ossification centers of the head and trochanters. At operation the neck of the femur was exposed and an 18-gauge needle was inserted into the cystic cavity through the periosteum and the medial cortex. Gentle aspiration yielded three cubic centimeters of a viscous, straw-colored fluid. After unroofing the cavity, four cubic centimeters more was obtained. The fluid did not coagulate on standing. The cavity was unilocular and was lined by a thin grey membrane which was curetted. The cavity was then filled with bone-bank bone chips. Histological examination of the membrane revealed typical loose fibrous tissue, with a lining layer of flattened cells resembling fibroblasts and numerous thin-walled endothelial-lined vessels. The case is too recent for follow-up report.

In all cases a specimen of venous blood was drawn shortly after the surgical

procedure and subjected to the same analysis as the cyst fluid. The values for individual determinations, which are tabulated in Table I, were obtained after the coagulated specimens of blood and fluid had been centrifuged to spin down the clot. In each case only one fluid specimen was analyzed. In all instances the supernatant fluid in the centrifuged sample of cyst fluid and blood was clear and straw colored. The clot which was spun down from clear specimens of cyst fluid resembled fibrin in appearance, but chemical analysis for fibrin was not attempted. With the qualitative acetic acid test no mucin clot appeared in any of the specimens.

DISCUSSION

Judging by gross inspection, the cysts contained either of two types of fluid—one resembled blood, the other serum. In three patients (Cases 3, 5, and 6) the fluid was serous and in another two (Cases 1 and 2) it was sanguineous. One of the latter two patients (Case 1) had had two previous surgical procedures; at the first two the fluid was serous but at the third procedure the cyst fluid resembled blood. The other case (Case 4) was of interest in that the cyst fluid was serous at first and, despite continuing gentleness during aspiration, it became admixed with blood, and finally was entirely composed of blood. All of the patients gave the history of one or more fractures through the cyst. The fluid samples from all of the patients had a concentration of protein very similar to that found in the patients' venous blood. All samples of fluid, except that in Case 6, coagulated on standing. All of the fluids resembling blood had hematocrit values near 40 per cent. The chemical determinations listed in Table I bear out the general similarity between cyst fluid and serum.

Can the characteristics of the cyst fluids indicate their probable manner of formation? One way to study this question is to compare the fluid with other body fluids. Because of its high protein content, the cyst fluid differs greatly from spinal fluid, tears, sweat, and urine and resembles either serum or those pathological accumulations of fluid which would be expected to have a large admixture of serum, for example, fluids from subdural hematomata[5] and hygromata, effusions into joints or other body cavities as a result of inflammation or neoplasm.

The electrophoretic patterns of normal and pathological body fluids recently have been summarized and discussed by Owen. The protein components in the normal fluids other than serum usually have to be concentrated by one procedure or another before determinations of pattern can be done. Even after this possible source of error is introduced, most normal fluids broadly resemble serum in pattern. The normal mean values and normal range for serum as compiled by Owen have been listed in Table I. Also listed are his estimates of what might be considered mild or moderate changes in these values. From a comparison of Owen's data with the determinations obtained in this study, it is apparent that there is no great deviation in any one of these determinations on cyst fluid from the normal range in serum. Owen's account also records the demonstration in other body fluids (bile, urine, and spinal fluid) of protein components not found in normal serum. However, the methods used for the demonstration of these components were far more elaborate than those used in this study, and the possibility still remains that cyst fluid may contain protein or other components not found in serum.

Synovial fluid merits comparison with cyst fluid because it contains significant amounts of protein (1.07 to 2.13 grams per 100 cubic centimeters according to Ropes and Bauer, 1953). Other accounts of synovial-fluid protein determination have been recorded with much higher concentrations which resemble the concentrations found in the cyst fluids described here. One study[3] recorded values of 3.6 to 6.3 grams per 100 cubic centimeters and another[1], values 1.1 to 3.3 grams lower than the total serum protein. However, in both of these studies, the fluids used were obtained from joints after trauma or from sterile but presumably in-

flamed joints, in contrast to the studies of Ropes and Bauer, which were on fluid from normal joints.

As expected, because of their small particle size, the concentrations of electrolytes and non-protein nitrogen in synovial fluid, cyst fluid, and serum are the same. The presence of mucin in synovial fluid and its absence in the cyst fluids is to be noted. The absence of fibrinogen or fibrin in synovial fluid and its probable presence in the cyst fluids, at least in the five which coagulated on standing, should also be noted. All of these data lead to the conclusion that the cyst fluids do not bear any resemblance to synovial fluid, despite the histological similarity of the lining cells and subjacent tissues of the cyst walls to synovial membrane. Before this study was begun, a relationship between cells of the synovial membrane and the lining cells of cysts was suspected on morphological grounds. In an unpublished case several small cavities were found in the membrane lining the principal cyst cavity. The lining of the small cavities was similar to that found in most cysts — flattened fibroblastic cells — and the fluid within the small cavities was stained light pink with hematoxylin and eosin. The possibility that cyst fluid may be produced by secretion rather than diffusion or, at any rate, may collect in several small cavities rather than in a single one becomes an important consideration in pathogenesis. The case with multiple cavities is unusual, however, since the majority of cysts are unicameral. Only in a few are there non-osseous septa lined with flattened fibroblastic cells. Jaffe and Lichtenstein, emphasizing the unicameral aspect of the bone limits to simple cysts, stated that fibrous or soft-tissue septa may divide the cavity. They attribute these septa to repair after trauma. As indicated in Case 1 it is evident that the trauma of fracture or of surgery can compartmentalize a cyst by bone as well as by fibrous septa. The interpretation that fibrous septa can arise only as a result of trauma does not seem to be adequate in view of the absence of histological evidence of a reaction to trauma in the septa. The lack of any resemblance of cyst fluid to synovial fluid also discourages etiological speculation in terms of a secretory mechanism, that is, a dysplastic process in which the involved connective-tissue cells assume synovial potentialities. The significance of the existence of a single chambered cavity or of a cavity with one or more partitions will become apparent from another point of view when a second possible mechanism of formation of cyst fluid—from interstitial fluid—is considered.

Interstitial fluid contains less protein than does plasma. Gitlin[6] calculated that the concentration of albumin in interstitial fluid is 0.7 to 0.9 gram per 100 cubic centimeters, as shown by iodinated albumin studies with I^{131}. Lymph from unobstructed lymphatic vessels resembles interstitial fluid with respect to protein concentration and composition. Gitlin stated that only when there is obstruction to lymphatic flow with resultant accumulation of lymph or where lymph drainage is not available to a tissue space (for example, certain cysts and subdural hematomata[5]) can the protein concentrations of the enclosed fluid approximate those of the serum. If we apply this concept to the formation of bone cysts the first difficulty we encounter is conceiving what may be the initial lesion. It has never been established that the formation of a bone cyst is preceded by any recognizable pathological change, traumatic or otherwise. In fact, any cyst, which is recognized as such, is a rather large lesion when first encountered. The unique anatomical distribution and age incidence of the lesion point to the possibility that some disturbance in the normal development of the metaphyses allows for the development of the fluid-filled cavity. What anatomical variation could permit a relatively rapid accumulation of fluid?

In the bones of children the metaphyses are the sites of most rapid remodeling of bone. The remodeling is the result of a composite of resorption and deposition of bone; the resorptive process, when very rapid, is often accompanied by the formation of foci of a loose fibrous tissue which resembles that seen in the wall of cysts. This tissue is a well known accompaniment of nearly all physiological and

pathological processes where resorption is very rapid, as in hyperparathyroidism and renal osteitis fibrosa. On a microscopic scale this fibrous tissue may be observed in histological preparations from many sections of the metaphyses of normal growing children. One or more of these foci of fibrous tissue, according to the concept presented here, represents the first step in cyst formation.

For the second step, the accumulation of the cyst fluid, consideration must be given to the movement of fluids in cancellous bone of the metaphysis. In this area the vessels present are extremely thin-walled sinusoids. Very little is known concerning the intimate circulation of blood and interstitial fluid in this locality, but it is said that the blood flow is extremely slow and the blood pressure is very low. The unique vascular arrangement, where arteries, capillaries, and veins are absent and the only vessels present are sinusoids, prohibits measurement of intravascular hemodynamic pressures and discourages analogies with such measurements in soft tissues. The added fact that lymphatic channels are reported not to occur in the bone marrow thus raises the question as to how drainage of interstitial fluid from the marrow spaces occurs. In Gitlin's study the dynamic aspects of flow of interstitial fluid into the vascular system and the interchange of plasma proteins between the interstitial fluid and the plasma are considered. Since interstitial fluid has a much lower protein concentration than plasma, the net return of the protein back to the vascular system can occur only by way of lymph channels. The interchange of water between interstitial fluid and plasma however can occur without the mediation of the lymphatic system. With these factors in mind, the formation and continued existence of a bone cyst may be predicated upon a blockage in the drainage of interstitial fluid within the metaphysis. The blockage would have to be of long enough duration to allow not only the collection of a significant amount of protein, and hence interstitial fluid, within the cancellous tissue but also a sufficient period of time for that fluid to equilibrate with the serum. The blockage would also have to continue for the length of time necessary to cause resorption of large numbers of the bone trabeculae traversing the affected drainage bed. The increase in pressure of interstitial fluid would not have to be very great to exceed the venous pressure, nor would this slight rise in interstitial fluid pressure yield evidence of markedly increased pressure when the cysts were entered. The pressure findings in the cases under discussion here would therefore be explained by this scheme.

The site of the accumulation of fluid might well be an area of interstitial tissue in which the sinusoid vessel was occluded by trauma and thrombosis. Another possibility is that one of the branches of the sinusoid vessels, in proliferating at the face of the epiphyseal cartilage or in the rapidly remodeling metaphyseal zone, might be formed without an adequate opening into the vascular system—that is, it might be wholly or partially blocked. Were either method of blockage to occur, fluid would collect in the space. Although its initial protein content would be that of interstitial fluid, in time the fluid would equilibrate across tissue septa with fluid in vessels which are not blocked and would increase its protein concentration to that of the plasma. Gitlin calculated that equilibrium between the plasma fraction of protein and the extravascular fraction normally occurs by diffusion in about two days. With the conditions envisioned, the equilibrium of protein between cyst fluid and plasma would take much longer. However, as long as the hydrodynamic pressure within the space exceeds that of the surrounding well drained interstitial tissue, the space would enlarge. Since the hemodynamic pressures in this region normally are very low, only a small increase in pressure of the blocked area would be necessary for continuing expansion of the cyst. Expansion would cease when collateral drainage became effective, or when blockage was relieved. Thus, a cyst in which the vascular blockage was relieved might very well show spontaneous resolution, and a cyst whose collateral drainage became effective might persist without further expansion while normal ossification at the epiphyseal line was resumed.

The occasional demonstration of blood rather than serous fluid in the cyst cavity may be explained by trauma. The one patient in whom gentle aspiration induced hemorrhage into the cyst (Case 5) indicates that the traumatic force need not be great. The original presence of serous fluid in five of the six cases under discussion indicates that this type of fluid is the more usual if not the universal type of fluid originally present. The persistence of blood in the cavity may also depend on blockage of the interstitial-fluid drainage pathways but the vascular arrangements and pathways for drainage of the blood-filled cavity would differ somewhat from those of cysts with serous fluid. Here a connection with the vascular tree, however small, would probably have to be postulated to account for the blood.

TABLE I

Patient *	Na	Sugar	Cl	NPN	TP	Electrophoresis (*Per cent*)				
						Albumin	Alpha[1]	Alpha[2]	Beta	Gamma
L. W. CF	144	146		28	6.9	65	2	6	14	13
VB	140	99		48	7.0	67	3	5	14	11
E. T. CF	149		103	19	6.6	58	4	12	11	14
VB	146		102	26	7.3	54	5	12	10	19
W. L. CF	143		100	24	5.2	60	5	6	12	18
VB	145		104	29	6.4	54	4	12	15	15
F. L. CF	141	100		21	5.4	74	3	4	8	11
VB	139	126		24	4.9	69	3	5	10	13
J. C. CF	140		106	30	5.3	53	7	14	14	12
VB	157		108	38	7.4	63	5	8	14	11
G. D. CF	136	117	104	21	5.5	62	4	7	9	18
VB	136	194	98	32	6.5	66	2	11	9	12
Normal serum mean (Owen)						60	4	8	12	16
Normal serum range (Owen)						52–68	2–6	5–11	8–16	10–22
Slight change (Owen)						44–52	6–8	11–14	16–20	22–28
Moderate change (Owen)						28–44	8–12	14–20	20–28	28–40

* CF is cyst fluid; VB is venous blood obtained at the same time the cyst was aspirated.

Even an occasional instance of multiple compartments in a lesion requires a more complex speculative account of pathogenesis than if all bone cysts were unicameral. One might suggest dilatation of several rather than one tissue space (for example, sinusoids pinched off from the vascular tree, or lymph spaces, or, as in the present theory, pathologically large tissue spaces). Alternative possibilities, the conversion of a single space into compartments by fibrous reaction, or amalgamation of several spaces into one large one, also are possible.

The present theory of pathogenesis, as well as all of the others advanced, has not been able to explain at least one rather obvious characteristic of bone cysts: their predilection for certain localizations such as the upper end of the humerus. As outlined, the present concept envisages an unusual obstacle to drainage of interstitial fluid. Perhaps uncommon anomalies in the development of vessels of microscopic size, with or without the influence of trauma, may be necessary to supplement the theoretical account to explain the localization of these lesions. Certainly trauma alone, as a frequent cause of thrombosis, is an insufficient etiological factor, or else the cysts would develop in more exposed sites.

The lack of bone trabeculae within the cyst also needs explanation. Presumably, during the development of the cyst, trabeculae are resorbed. Since markedly increased pressure of cyst fluid has not been demonstrable, the bone

resorption has to be attributed either to a postulated slightly elevated hydrodynamic pressure within the cavity, or to changes in composition of the now stagnant fluid bathing the trabeculae—changes from the normal interstitial-fluid composition which render it indistinguishable from serum.

While it is true that this theory rests on a number of unproved speculations, it nevertheless can explain many of the known phenomena concerning cysts: the type of fluid contained, the type of tissue reaction in the cyst membrane, the pressure of fluid in cysts, and most of the clinical characteristics of cysts. In these respects it compares very favorably with other theories of etiology and pathogenesis which have been reviewed by Garceau and Gregory and need not be repeated here. The present theory also has the advantage of indicating avenues of investigation of this lesion which may lead to further clarification if not to more adequate treatment. The availability of tracer iodinated plasma proteins which may be injected into cysts for investigation of the problem just discussed or injection of radiopaque media for this same purpose may yield valuable data.

SUMMARY

Cyst fluid of six simple cysts of bone was aspirated and determinations of electrolytes and protein content made. In four of the cysts the fluid resembled plasma. In two others it resembled blood. A theory of etiology of simple cysts is suggested in which the principal etiological factor is blockage of the drainage of interstitial fluid in a rapidly growing and rapidly remodeling area of cancellous bone.

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Vascular Patterns in Immobilized, Denervated, or Devascularized Rabbit Limbs*

BY ALBERT B. FERGUSON, JR., M.D., AND YOSHIHIKO AKAHOSHI, M.D., PITTSBURGH, PENNSYLVANIA

From the Department of Orthopaedic Surgery, University of Pittsburgh Medical Center, Pittsburgh

The atrophy of skeletal muscle secondary to immobilization appears to differ from the disuse atrophy caused by denervation or tenotomy in that it appears to be influenced by the tension of the immobilized muscle. The completely relaxed muscle shows marked atrophy, but the moderately stretched muscle appears much less subject to weight loss and, at times, may even hypertrophy [1-5]. The changes in chemical composition that take place with atrophy have been shown by one of us [2] to be more than a mere change in water content.

In an attempt to understand some of the physical forces that influence the metabolism of muscle after the muscle has been immobilized, denervated, or devascularized, arterial injection studies were performed on muscles subjected to these aberrations. These studies are not a measure, of course, of either the rate or volume of blood flow in the muscle, but, nonetheless, the changes that were observed with this technique may help in understanding what happens to muscles in an immobilized, denervated, or devascularized limb.

FIG. 1

In this animal, immobilized by cast for six weeks, dilatation of the major arteries of the right lower extremity is shown (on the reader's left).

MATERIALS AND METHODS

A total of forty-eight male and female rabbits weighing between 1,800 and 2,500 grams was divided into six groups for the purpose of this study.

In Group I the right limb was padded with sheet wadding and a long-limb plaster cast was applied with the knee at 90 degrees of flexion and the foot in neutral position at right angles to the axis of the tibia. In Group II two pins were inserted transversely, one in the supracondylar region of the right femur, the other in the proximal diaphysis of the tibia. A plaster cast was then applied, incorporating the protruding ends of the pins and holding the limb in the same position as in Group I. In Group III one or more pins were placed across the

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FIG. 2

After six weeks of immobilization by skeletal pins without cast fixation, the dilatation of major arteries in the animal's right lower extremity is not so evident, although the limb generally is hypervascular as compared with the animal's left lower extremity.

knee and ankle joints, thereby securely fixing the limb in the same position as in Groups I and II. The pins were cut off beneath the skin, and the limb was not immobilized in any other way. In Group IV the sciatic nerve was divided at the level of the tip of the greater trochanter, and a segment 0.7 centimeter in

FIG. 3

Six weeks after tenotomy of the tendo achillis, the major arteries appear similar except for some straightening of the posterior tibial artery. There is some local hypervascularization superior to the os calcis in the region of the operative trauma (on the reader's left).

length was resected. In Group V, tenotomy of the tendo achillis was performed on the right side, with resection of 1.5 centimeters of the tendon. In Group VI the femoral artery was ligated by a silk suture as it passed beneath the inguinal ligament.

Three weeks after the experimental procedure, arterial injections through the abdominal aorta, under nembutal anesthesia, were done in half of the animals in each group before sacrifice. Six weeks after the original experimental procedure the animals in the remaining half of each group were injected and sacrificed in a similar fashion. As injection material, a warmed mixture of equal parts of a 1.5 per cent Berlin blue solution with 80 per cent suspension of barium (micropaque) was used. This proved to be a most successful injection fluid for muscle

FIG. 4

Ligation of the femoral artery increases the collateral circulation (as shown on the reader's left). The increased fine feathery hypervascularization of the muscles is also evident on comparison with the control side.

after numerous experiments with various combinations of a barium suspension, Berlin blue, and India ink had been tried. The usual amount used varied between twenty and thirty cubic centimeters. During injection the hip was held in 20 degrees of flexion and in a neutral position with respect to rotation, abduction, and adduction, while the knee was held in 20 degrees of flexion.

After the perfusion of approximately 20 cubic centimeters of the injection solution, a roentgenogram of the whole limb was made while the injection was continued under moderate hand pressure. After this roentgenogram the iliac artery was ligated, the skin was removed, and the limb was disarticulated at the hip and placed in 10 per cent formalin solution.

After fixation for ten days, the anterior tibial and gastrocnemius muscles were each dissected out, and roentgenograms were made. For some of the specimens roentgenograms were made, and then the limbs were decalcified and cleared by the Spalteholz technique to visualize the arterial pattern in three dimensions.

In all animals the normal contralateral limb and its muscles were subjected to the same procedures as used for the immobilized extremity, so that each animal served as its own control.

RESULTS

Changes were observed in these limbs in the main arteries, in the small vessels about the knee and the ankle, and in the vessels within the gastrocnemius and anterior tibial muscles.

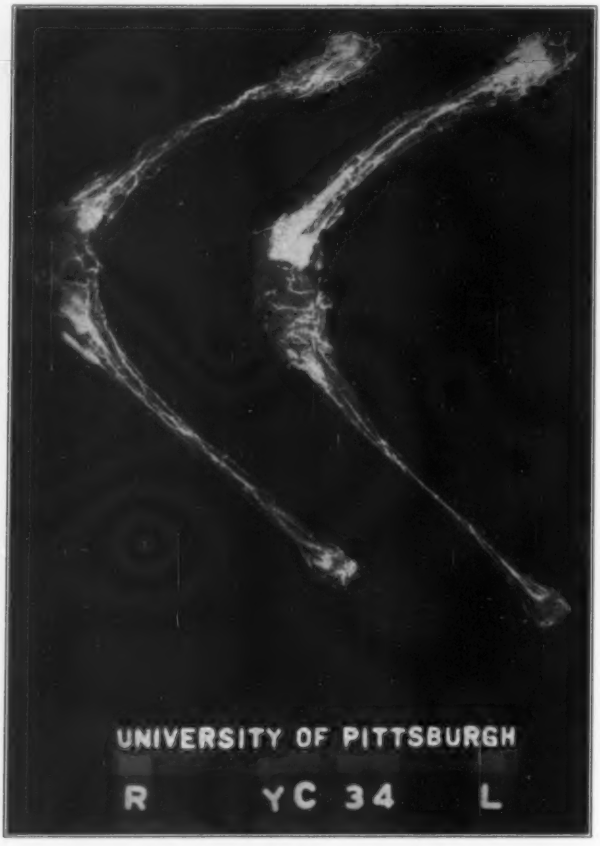


FIG. 5

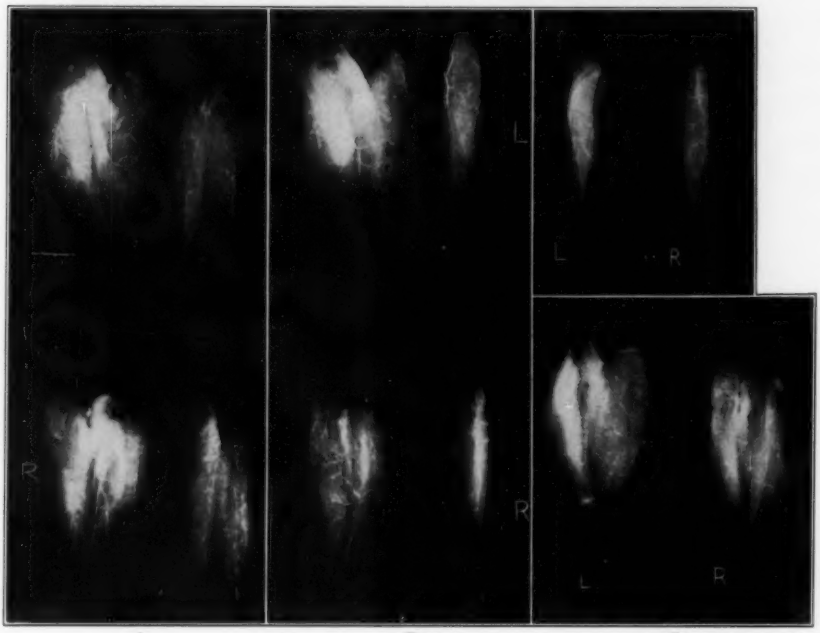
In this preparation, with only the periosteum and soft tissues about the knee and ankle joints remaining, the hypervascularization occurring at the knee and ankle when the animal has been immobilized by a cast for six weeks is shown on the animal's right side.

Changes in the Main Arteries

In the limbs immobilized in plaster casts (Group I) some dilatation was observed in all specimens. In a few, the dilitation was extreme but in about one-third of the limbs in this group vascular dilatation was so slight that it was barely appreciable. In Group II, in which both skeletal wires and a plaster cast were used, similar changes of the same degree were observed, but in the limbs with only internal fixation (Group III) no changes in the large arteries were observed. After division of the sciatic nerve (Group IV) there was no dilatation, but after tenotomy (Group V) minimum dilatation did occur. Ligation of the femoral artery (Group VI) also caused some dilatation.

Changes in the Small Arteries around the Knee and Ankle

In the first three groups there was increased filling of the small vessels around the knee and ankle, and more vessels were visible in these areas than in the control limbs. In the limbs in which skeletal wires were used for fixation this increased vascularity might be attributed to the presence of the pins. However, in Group I, in which only external fixation was used, increased vascularity occurred at both the knee and the ankle, and in Group II the ankle also showed increased vascularity even though no wires were placed near this joint. After division of the sciatic nerve (Group IV) these small vessels also showed increased filling, and there was an increase in the number of vessels visible. After tenotomy (Group V) there was increased filling of the small vessels at the ankle, which is to be expected as the result of tissue repair after tenotomy. However, the same vascular change was also very evident posterior to the knee and far from the operative site. After ligation of the femoral artery (Group VI) there was the development of collateral pathways but nothing particularly related to the joints *per se*.



A *B* *C*

FIG. 6

Roentgenograms of the anterior tibial and gastrocnemius muscles six weeks after immobilization in a cast (*A*), denervation (*B*), and skeletal fixation with pins across the knee and ankle joints (*C*).

A. The control muscles are above. No marked difference in the vascular pattern or size of the muscle mass has resulted after immobilization in a cast.

B. The diminution in the size of the muscle is apparent after denervation, as is the general decrease in the size of the experimental muscles (below).

C. The anterior tibial muscles, experimental and control, are above; the gastrocnemii, experimental and control, are below. Skeletal fixation for six weeks has not caused any appreciable change in the vascular pattern.

Vascular Changes within the Muscles

No changes in the vessels within the anterior tibial and gastrocnemius muscles were observed in Groups I, II, and III, in which the limbs were immobilized. However, after neurectomy (Group IV) a bizarre vascular pattern appeared, with fine hairlike vessels much smaller than normal. Similarly, after tenotomy (Group V) the vessels also presented a bizarre pattern of fine vessels — a pattern that appeared to be related to the change in the shape of the muscle caused by the

tenotomy. In the limbs in which there had been ligation of the femoral artery (Group VI) the vessels in the muscles showed the most striking change in the vascular pattern, being characterized by much greater than normal filling of minute vessels. This was particularly noticeable in the anterior tibial muscle, although it was also present in the gastrocnemius muscle. In all instances the vascular pattern was bizarre, with increased arborization of the vessels. Despite the striking vascular changes in all of these muscles, none of the animals showed any disability, and all their limb muscles appeared to be functioning normally prior to sacrifice.

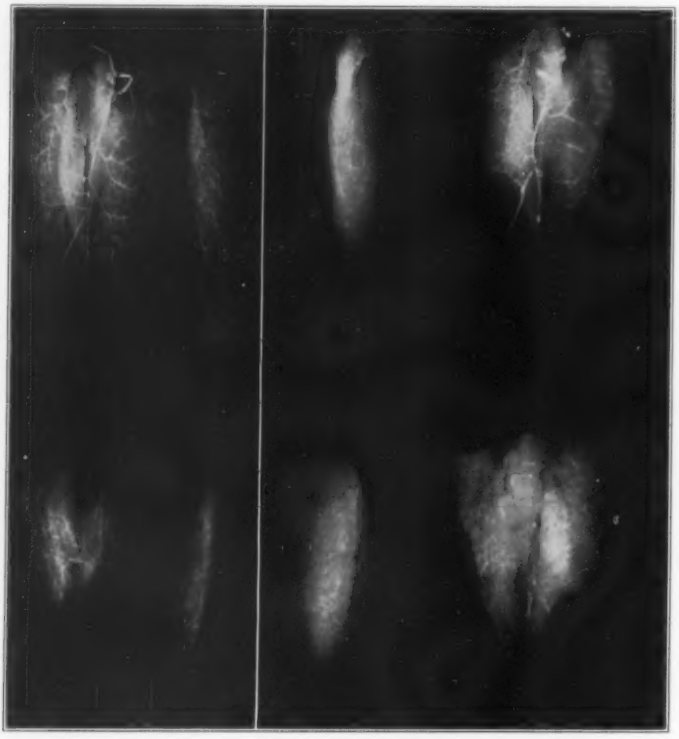


FIG. 7-A FIG. 7-B

Fig. 7-A: After tenotomy of the tendo achillis the experimental gastrocnemius and anterior tibial muscles (below) show loss of the normal vascular pattern and numerous fine longitudinal vascular channels.

Fig. 7-B: After ligation of the femoral artery the involved muscles (below) also show a marked increase in the numbers of vessels in the form of a feathering arborization of fine vessels within the muscle.

COMMENT

Several findings of significance appear to result from this study: (1) There was dilatation of the main arteries of the limbs that were immobilized in plaster casts with or without internal fixation that was not observed in the limbs in which internal fixation of a joint was used alone; (2) There was increased filling of the arteries around the knee and ankle when any form of immobilization was used; (3) There was increased arborization of fine vessels in the anterior tibial and gastrocnemius muscles even though the muscles otherwise appeared to be normal after ligation of the femoral artery; and (4) There was a change in the pattern and a decrease in the size of the vessels within the muscles after denervation or tenotomy.

In any study in which injection techniques are used, care must be taken to rule out artifacts caused by differences in positioning of the limbs or accidental differences in the injection pressure in the experimental limb as compared with the control. These sources of error appeared to be ruled out by the consistency of the results associated with cast immobilization. Examination of animals that had not had experimental procedures done to either limb uniformly revealed symmetrical results with this injection technique. One difference between the limbs immobilized in plaster and those in which internal fixation alone was effected was the fact that without the cast the joints of the limb which were not fixed internally could be moved even though the internal fixation kept the other joints in a constant position. Stretching of an artery previously immobilized may have had some mechanical effect on the vessel wall, thereby leading to dilatation. Similarly, immobilization may actually have weakened the wall so that dilatation of the vessel in the immobilized limbs may have occurred with pressure insufficient to dilate the vessels on the control side. Another possibility is that the artery may actually have been dilated by a greater volume of blood flow to the immobilized side; the presence of vascular enlargement does not prove this possibility, but this finding correlates well with the findings of previous methods that showed an increased blood flow to immobilized muscle, as discussed by Imig, Randall, and Hines in 1953 and Ferguson, Vaughan, and Ward in 1957.

It is apparent that the changes in vascular pattern around the joints that were immobilized are not related merely to cast fixation because similar changes were present when the joints were held by internal fixation above. This change in vascular pattern appears to be a definite finding and may correlate eventually with other changes found in joints after immobilization.

The increased arborization of the vascular pattern after ligation of the femoral artery, which was observed in the anterior tibial and gastrocnemius muscles, appears to be a definite change occurring in all four animals so tested. It was a unique change not to be confused with the pattern changes that followed denervation and tenotomy. A sympathetic trunk removal was done on the right side in six additional rabbits in which the limbs were injected in the same manner as described in this paper. No dilatation of the main artery or arborization of the muscle vessels was noted in any animals in this latter group. The increased arborization within the muscle appears to represent a change secondary to ligation of the main vessel in an animal with collateral vessels adequate to maintain the nutrition of the muscle.

SUMMARY

Using an injection technique, the vascular pattern in the hind limbs of rabbits was studied by means of roentgenograms. The right hind limbs of these animals were immobilized by plaster cast, by cast and skeletal pins, and by internal pin fixation of the ankle and knee joints. In addition, denervation, femoral-artery ligation, and tenotomy were done in other animals.

The changes in vascular pattern noted after cast immobilization were dilatation of the main arteries and increased vascularization about knee and ankle joints. After internal fixation alone similar changes occurred, except that there was no dilatation of the main arteries. Increased arborization of small muscle vessels was noted after femoral-artery ligation. Diminished vessel size and bizarre vascular patterns were noted after both denervation and tenotomy.

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DISCUSSION

Anterior Fusion of the Cervical Spine

(Continued from page 594)

inlay technique as the authors have demonstrated. Fusion also occurs when the bone graft is inserted into the intervertebral disc space as demonstrated by our own experience.

One must be very cautious about diagnosing instability or subluxation at one level of the cervical spine in those patients without an obvious history of fracture-dislocation. If, in flexion, one vertebra seems to slide forward on the one below, this may actually mean that the intervertebral level below the one that appears to sublux is splinted. Dr. Harris and Dr. Smith showed the importance of discography in choosing the level of disc abnormality when confronted with this diagnostic differential between two cervical levels of possible pathological conditions.

The cervical spine appears to fuse more readily than the lumbar spine, regardless of whether posterior or anterior fusion is performed.

Fracture-dislocation of the cervical spine due to the instability that may follow such injury very often gives a firm indication for early fusion. Sometimes, after such injury a spontaneous fusion occurs, but this is not the invariable course. When fusion does occur spontaneously either anteriorly or posteriorly the position of the cervical vertebrae may not be anatomical, but the postinjury pain often stops at the time of bone consolidation. Thus, the indication for fusion after an injury that leaves the cervical spine unstable has been indicated to us by this clinical observation of patients who have not had an operation for spine fusion but whose spines have fused spontaneously.

The problem of instability of the cervical spine at multiple intervertebral levels after extensive laminectomy, or at one intervertebral level after fracture-dislocation, is different from the problem of cervical-spine fusion at one or two intervertebral levels for disc degeneration, osteo-arthritis, and nerve-root pain, particularly in regard to postoperative care. When instability is a major consideration, about six weeks of prolonged recumbency after operation appears indicated.

Prolonged recumbency was used in Cases 1, 2, 3, 4, 7, and probably 5. In these patients, the final position of the cervical vertebrae was practically anatomical. All patients had several weeks of recumbency with traction or bracing in the postoperative period.

In Case 6, the patient was apparently ambulated early in a Minerva jacket, and a good deal of the preoperative deformity of the cervical spine seems to have recurred before the anterior fusion was solid.

Possibly, a Blount brace or some other mechanism for maintaining a head-neck elongation force after operation would be as adequate as recumbency after operation if a patient has to be ambulated in the immediate postoperative period.

The patient in Case 6 was relieved of pain by the fusion. However, both excellent anatomical and functional results are desired, and so I stress this point about postoperative recumbency when fusion is performed for marked instability — a point that Dr. Bailey and Dr. Badgley have, I believe, demonstrated in their case reports.

The surgical approach used by the authors appears relatively safe. Since using safeguards outlined by Dr. George Smith and myself (J. Bone and Joint Surg., **40-A**: 607–623, June 1958) for the anterior approach to the cervical spine we have had no complications. No intraspinal neurological complications have occurred in any of our fifty-one patients.

The principles and practice of cervical-spine surgery are gradually evolving. The work of Dr. Bailey and Dr. Badgley presented here is a solid addition to this body of surgical knowledge.

Blood Supply of the Human Tibia*†‡

BY GEORGE E. NELSON, JR., M.D., PATRICK J. KELLY, M.D., LOWELL F. A. PETERSON, M.D., AND JOSEPH M. JANES, M.D., ROCHESTER, MINNESOTA

From the Section of Orthopaedic Surgery, Mayo Clinic and Mayo Foundation, Rochester

Following Langer's early and extensive monograph on the circulation in bones, many investigators have studied the problem. It is generally conceded that three main systems of vessels supply the long bones: namely, (1) the epiphyseal-metaphyseal; (2) the nutrient (*arteria nutricia tibiae*); and (3) the periosteal. Beyond this point harmony ceases. The main areas of contention are: (1) the relative importance of the periosteal vessels; (2) the distribution of the venous system; (3) the type and number of vessels in the Haversian canals; and (4) the question of whether vessels of growing animals cross the epiphyseal plate. Since all our specimens were obtained from adult human beings, we will not consider the last problem.

Although Lexer and his associates made no mention of the periosteal circulation in their monograph, most workers have stressed the importance of the periosteal vessels [11,13,16]. Johnson believed that periosteal vessels supply only the outer half of the cortex. In more recent studies, however, Macnab, Brookes and Harrison, and Brookes questioned the importance of the periosteal circulation.

Concerning the arrangement of the venous system within bone, de Marneffe and Morgan each maintained that the veins are completely separated from the arteries, whereas Tilling and Weinmann and Sicher stated that the veins and the arteries accompany each other. A review of standard textbooks of histology [1,4,8,17,22,24] also failed to reveal any agreement as to the type and number of vessels contained within the Haversian systems. Some of this lack of agreement, we think, is due to species variation in the specimens studied. We hope in this paper to supply the answer to some of these controversial questions.

METHOD

Material for our study was derived from fourteen limbs that were amputated because of malignant lesions of the femur or soft tissues of the thigh. Several methods of study were utilized in the belief that a more accurate picture could be obtained by the employment of different approaches to the problem. Twelve of the fourteen specimens were studied by gross dissection. India ink was injected into the arterial trees of ten of the limbs that were then cleared by the Spalteholz technique [23]. Three limbs were examined by microangiography, after injection with micropaque§. In one limb a mixture of India ink and micropaque was injected so that both microangiography and the Spalteholz method could be used on the same specimen. Finally, sections were obtained from four tibiae for histological study; these were decalcified, embedded in celloidin, and stained with hematoxylin and eosin. The details of these investigative methods were described previously by other workers from the Mayo Clinic [5,9,10,20,21].

RESULTS AND OUR INTERPRETATION

In our specimens the tibial blood supply was found to be derived from three

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§Damancy and Company, Ltd., Ware, Herts., England.

FIG. 1-A

Transverse section through the proximal epiphysis of the tibia. The vessels enter from all sides like the spokes of a wheel (Spalteholz preparation using India ink, reduced slightly from × 2).

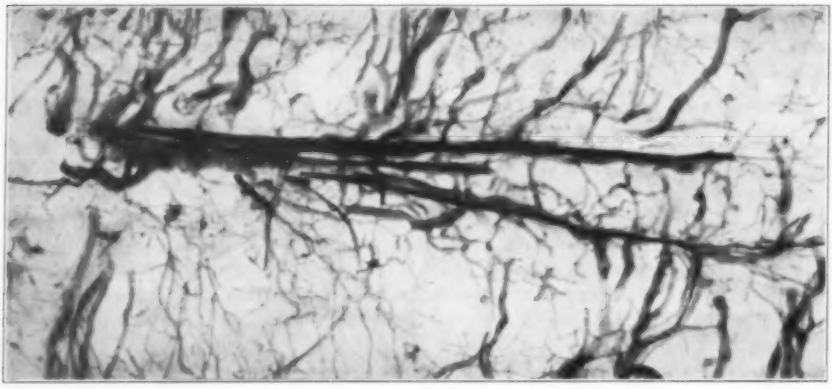

FIG. 1-B

Individual vascular spoke from the epiphysis. It is composed of several arteries and veins. The arteries appear black, and the veins appear gray (Spalteholz preparation of India ink and micropaque, reduced slightly from × 8.3).

main systems: (1) the epiphyseal-metaphyseal; (2) the nutrient; and (3) the periosteal. Although these systems will be discussed separately, we believe that they are closely interrelated, and we shall attempt to prove this point as we proceed.

Epiphyseal-Metaphyseal Vascular System

In the region of the epiphysis and the metaphysis of the tibia we found numerous vessels, indicating a copious blood supply. A prominent vessel in the proximal

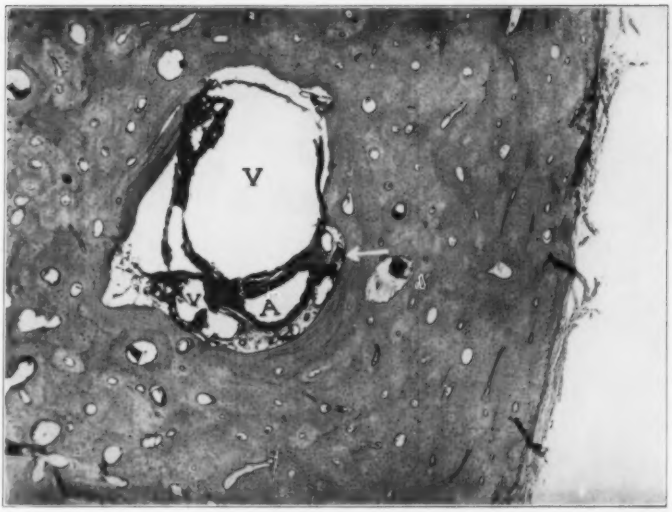

FIG. 2-A

Nutrient artery and two veins in the nutrient canal (hematoxylin and eosin, × 30). (A small nerve is indicated by the arrow.)

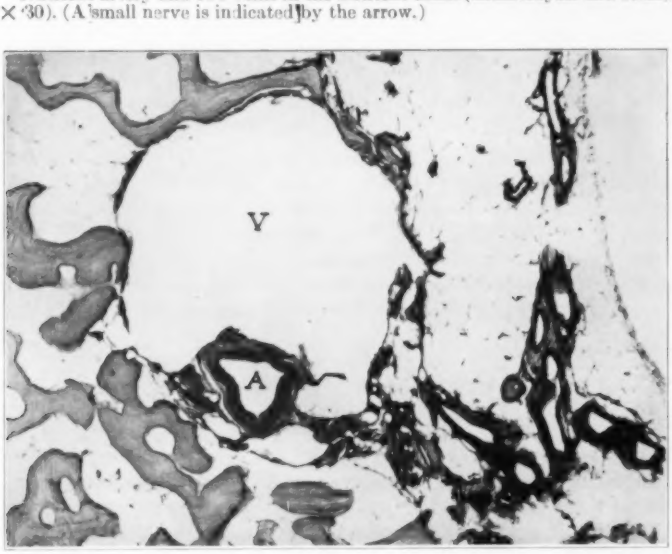

FIG. 2-B

Nutrient artery and one vein just after entering the medullary portion of the tibia. Note the ascending branches of the nutrient vessels to the right (hematoxylin and eosin, × 30). (*V* = vein; *a* = artery.)

part of the tibia originated from the middle genicular artery and entered the intercondylar eminence to supply the posterior portion of the epiphyseal region primarily. Also in this area were numerous vessels that entered from the periphery of the epiphysis like the spokes of a wheel (Fig. 1-A). The majority of these vessels appeared to originate from branches of the inferior genicular artery and tibial recurrent artery. Each spoke was composed of several arteries and their accompanying veins (Fig. 1-B). From each spokelike vessel many branches came off at right angles to form a dense, interlocking vascular network. Coming

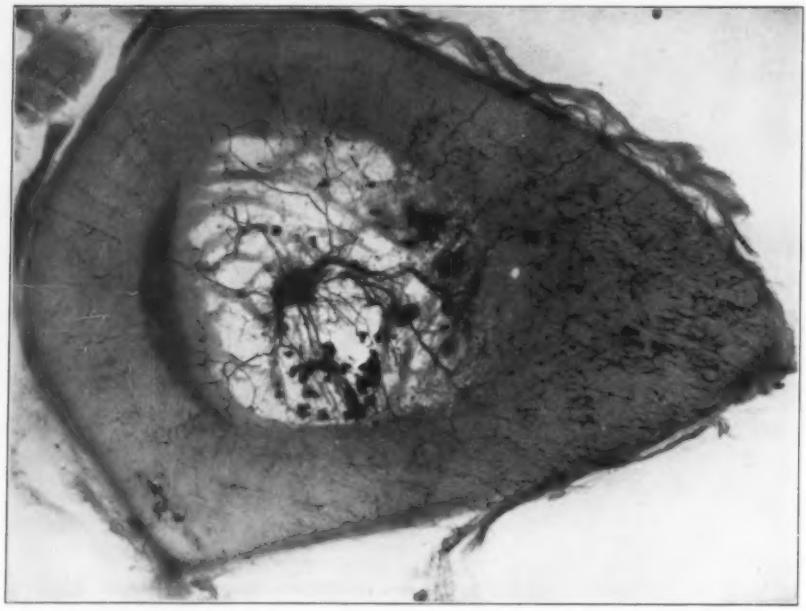

FIG. 3-A

At this level (mid-shaft) the single nutrient artery is seen located centrally. Radial arterial branches pierce the endosteal surface of the cortex (Spalteholz preparation, × 5.2).

up from below into the proximal metaphysis were terminal twigs of the ascending branches of the nutrient artery that anastomosed with the metaphyseal vessels. In the metaphyseal area of the distal part of the tibia a similar spoke-and-wheel arrangement of vessels was found. Particularly dense collections of vessels were noted in the region of the medial malleolus and also in the vicinity of the inferior tibiofibular joint. The vessels in the distal metaphysis were also linked with branches of the nutrient vessels that descended through the marrow cavity.

Nutrient Vascular System

In the twelve limbs that we dissected, the nutrient artery of the tibia consistently arose from the posterior tibial artery and penetrated the posterolateral cortex at a point just below the oblique line of the tibia, the origin of the soleus muscle. After entering the cortex of the tibia, the vessel coursed diagonally through the cortical bone for approximately five centimeters before it entered the marrow cavity. In our specimens we did not observe any branches originating from the nutrient artery as it passed through the cortex (Fig. 2-A). Within the nutrient canal, the artery was accompanied by two thin-walled veins and a myelinated nerve, whereas in the medullary cavity each arterial stem had only one adjoining vein. After emerging from the endosteal surface of the cortex, the nutrient artery quickly divided into several (usually three) ascending branches (Fig. 2-B). A single major descending branch continued across the medullary canal to lie close to the endosteal side of the medial cortex of the tibia. Lower down this vessel again became centrally (Fig. 3-A) located in the medullary cavity and finally split into several smaller vessels. The ascending and descending arteries in the marrow cavity gave off smaller branches that proceeded radially to pierce the endosteal surface of the cortex and then subdivided into small vessels that supplied the vessels in the Haversian canals. In some instances when the India-ink-injection technique was employed, we were able to demonstrate a vein accompanying an artery out into the cortex (Figs. 3-B and 4).

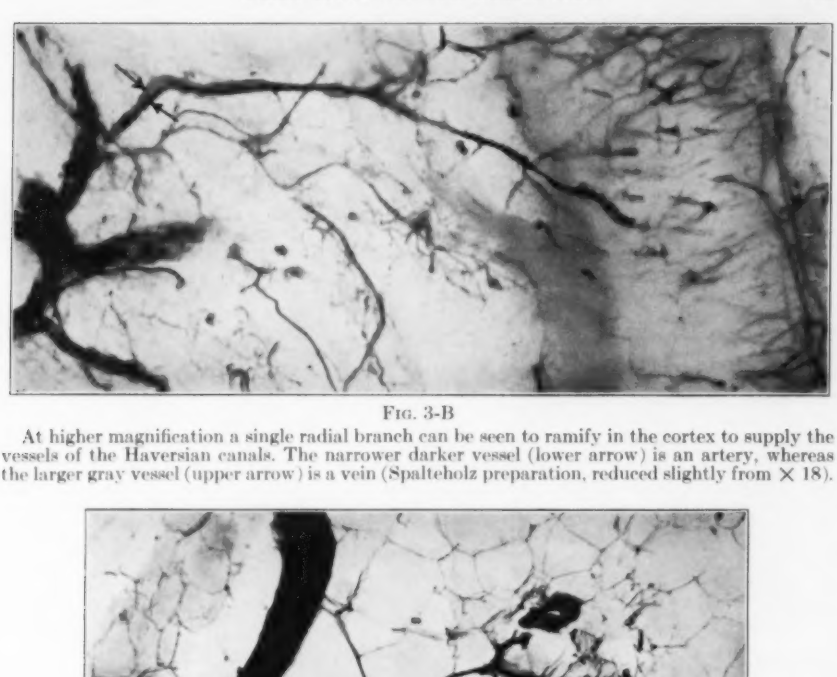

FIG. 3-B

At higher magnification a single radial branch can be seen to ramify in the cortex to supply the vessels of the Haversian canals. The narrower darker vessel (lower arrow) is an artery, whereas the larger gray vessel (upper arrow) is a vein (Spalteholz preparation, reduced slightly from × 18).

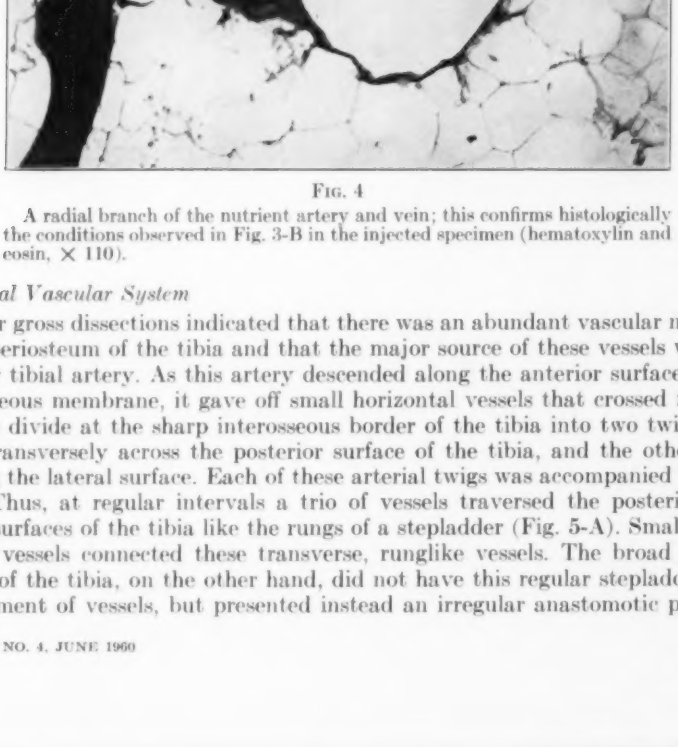

FIG. 4

A radial branch of the nutrient artery and vein; this confirms histologically the conditions observed in Fig. 3-B in the injected specimen (hematoxylin and eosin, × 110).

Periosteal Vascular System

Our gross dissections indicated that there was an abundant vascular network in the periosteum of the tibia and that the major source of these vessels was the anterior tibial artery. As this artery descended along the anterior surface of the interosseous membrane, it gave off small horizontal vessels that crossed medialward to divide at the sharp interosseous border of the tibia into two twigs, one going transversely across the posterior surface of the tibia, and the other twig crossing the lateral surface. Each of these arterial twigs was accompanied by two veins. Thus, at regular intervals a trio of vessels traversed the posterior and lateral surfaces of the tibia like the rungs of a stepladder (Fig. 5-A). Small longitudinal vessels connected these transverse, runglike vessels. The broad medial surface of the tibia, on the other hand, did not have this regular stepladder-like arrangement of vessels, but presented instead an irregular anastomotic pattern.

FIG. 5-A

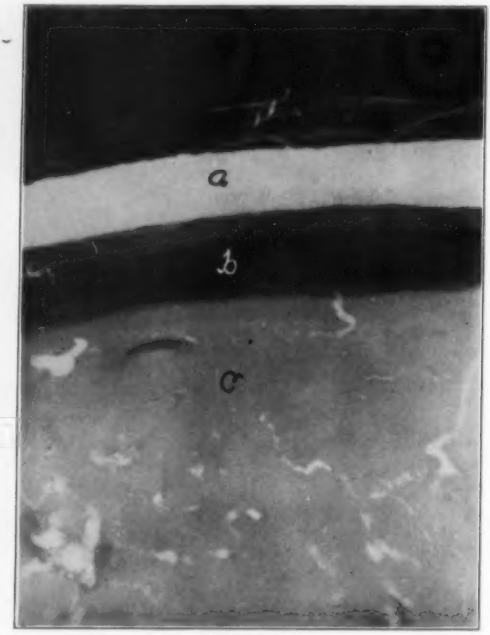

FIG. 5-B

Fig. 5-A: Trios of periosteal vessels on posterior surface of tibial shaft (gross specimen).
Fig. 5-B: The central artery of the trio of vessels demonstrated in Fig. 5-A. No arterial branches were seen to enter the cortex from these arteries (microangiogram, × 30). (*a* = artery; *b* = the inner layer of periosteum; *c* = cortical bone.)

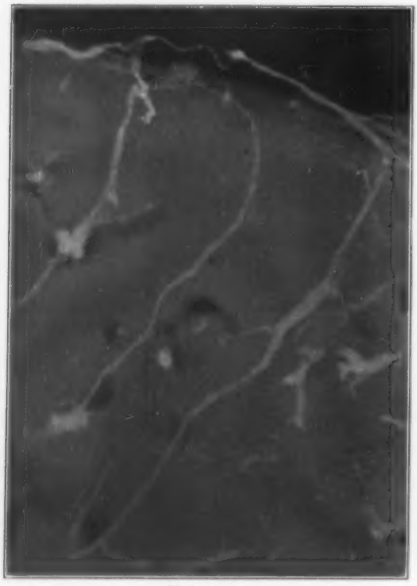

FIG. 6-A

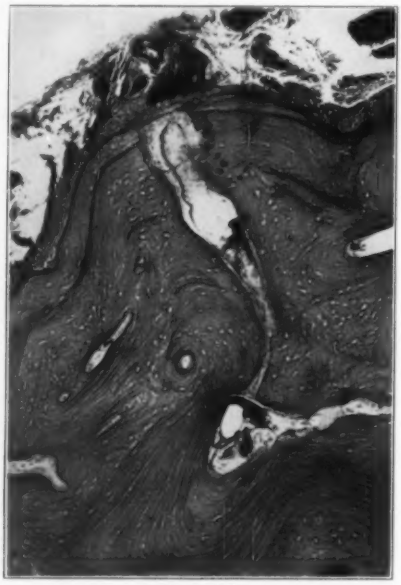

FIG. 6-B

Fig. 6-A: Vessels extending from the periosteal surface to endosteal surface (microangiogram, × 27).
Fig. 6-B: Microscopically, the vessels demonstrated in Fig. 6-A appear to be veins (hematoxylin and eosin, × 90).

Importance of Periosteal Vessels to the Blood Supply of the Cortex

In our material we were unable to demonstrate any arterial branches that arose from the central arteries of the trios of vessels in the periosteum and entered the cortex of the tibial shaft (Figs. 5-A and 5-B). In some of our cleared sections, on the other hand, and in some of the microangiograms especially, we were able to demonstrate in the diaphyseal cortex vascular channels that extended from the periosteal to the endosteal surface (Figs. 6-A and 6-B). These channels contained only relatively large tubelike vessels, the walls of which were composed solely of endothelium. A second and smaller type of vessel (seen only in our microscopic preparations) was also observed to arise from the small arterioles

FIG. 7

A thin-walled tube composed of endothelial cells can be seen entering the cortex from the periosteal surface; this appears to come from small arterioles at the juncture of the periosteum and cortex (hematoxylin and eosin, × 275).

that lie in nests in the periosteum and enter the cortex (Fig. 7). Microscopic study of sections stained with hematoxylin and eosin showed that all these vessels, entering or leaving the periosteal surface of the diaphysis, had varying diameters and thin walls lined by endothelium.

We believe that the larger vessels in the cortex (Fig. 6-B) provide venous drainage, whereas the smaller vessels (Fig. 7) are capillaries entering the cortex from the periosteal arterioles. This theory is bolstered by the fact that we could find veins but no arteries in the periosteum at the points where the larger cortical vessels made their exits, whereas we usually did find a nest of small vessels containing at least one small artery or arteriole at the sites where the smaller cortical vessels penetrated the periosteal surface of the cortex. We should like to empha-

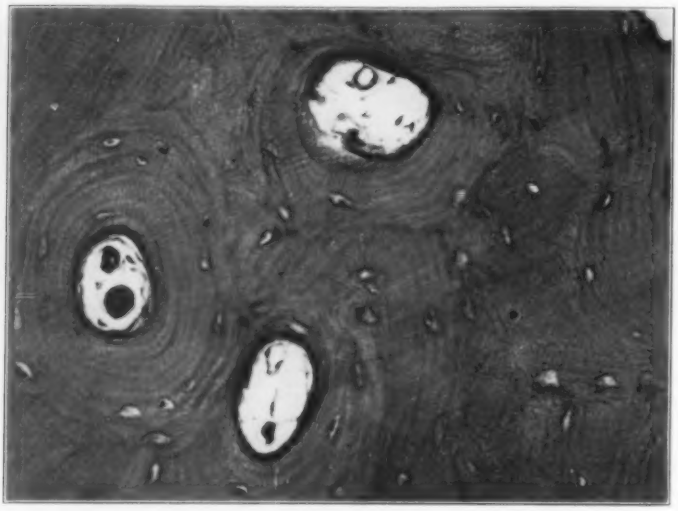

FIG. 8-A

Typical Haversian canals with two capillaries in each canal (hematoxylin and eosin, × 350).

FIG. 8-B

Single arteriole and what is probably a thin-walled vein in the Haversian canal. A small nerve, which is indicated by the arrow, is also seen. This arrangement is distinctly unusual (hematoxylin and eosin, × 400).

size, however, that although these capillary openings could be found in some areas at regular intervals, they were by no means numerous and probably represent no more than a potential source of collateral circulation that would function only if the principal blood supply is reduced. The premise that the periosteal vessels that we have observed penetrating the cortex are veins and capillaries rather than arteries is supported by the fact that our India-ink sections failed to demonstrate any extensive arborization of vessels from the periosteum within the outer part of the cortex. In histological preparations we could not see any vessels

entering the cortex from the periosteum that could be classified as arterioles.

In summary, therefore, we observed periosteal vessels of varying diameters entering or leaving the cortex, but these vessels always had thin walls that did not contain any muscle or elastic tissue. Thus, these vessels must represent either capillaries or small venules, but not arteries. From these observations we have concluded that in the normal adult human tibia the periosteal vessels usually play only a minor role in supplying arterial blood to the cortex, although the periosteal capillary bed is a potential source of blood supply for the cortex in the event of damage to the medullary blood vessels. This concept is supported by the experimental work of Trueta and Cavadias.

This theory of the role of the periosteal vessels in the cortical circulation does not imply that the periosteal circulation is not important. We believe that one of the main functions of the periosteal vascular system is to supply the vessels for new Haversian systems that develop on the external surface of the cortex—a theory that has been well documented by Ham. In our histological sections, in some areas we were able to find nests of vessels lying in grooves on the outer surface of the cortex. In other areas the osteogenic cells appeared to have proliferated around these grooves and to have enclosed the vessels so as eventually to form new Haversian canals.

Type of Vessels in Haversian Canals

Microscopic studies of stained celloidin-embedded sections of tibial cortex showed that the majority of the Haversian canals contained two or three vessels, but the number varied from one to four (Fig. 8-A). Many of the Haversian vessels were thin-walled structures resembling venules or capillaries, but other vessels with definite muscular walls were seen, and these are believed to be arterioles. We wish to emphasize, however, that the finding of vessels resembling arterioles in Haversian canals was definitely unusual (Fig. 8-B).

In general, it was noted that the Haversian canals tended to be larger near the endosteal surface and smaller near the periosteal surface. This observation was considered indirect evidence that arterial blood flow goes from the nutrient artery in a centrifugal direction from the endosteum to the periosteum, whereas the venous blood flows in the reverse direction from the periosteum to the endosteum and thence to the medullary veins. If this is the direction of flow, the Haversian canals near the endosteum would be larger than those near the periosteum since they have to accommodate the larger arterioles near the medullary artery and the larger venules resulting from the confluence of the venules of the Haversian systems throughout the cortex.

As already indicated, myelinated nerve fibers were observed in the nutrient canal, and on rare occasions small nerve fibers were seen in the Haversian canals in the sections stained with hematoxylin and eosin.

Venous System

At this point it might be well to recapitulate our findings with regard to the venous system. Other workers have mentioned the extreme thinness of the walls of the intramedullary veins, and our studies have corroborated this finding. We have noted that the walls of the veins—even those of the large nutrient vein—were only two or three cells in thickness with no muscular coat (Figs. 2-A and 2-B).

In the epiphyseal region where the vessels are arranged like the spokes of a wheel, we found that each spoke was composed of several arteries, each artery accompanied by one vein. Both the arteries and the veins in this spokelike arrangement gave off branches at right angles.

In the diaphyses the veins also accompanied the branches of the nutrient artery and even the small radial arterial branches to the cortex. In addition, there were irregularly located channels within the cortex—channels that contained vessels that we believe were veins draining blood to the periosteal surface. Within

the nutrient canal the artery was accompanied by two veins, but within the medullary cavity only one vein adjoined each artery. Some authorities prefer to call the thin-walled nutrient vein a "sinus" but to us this is purely a matter of preference in terminology. In some specimens the nutrient vein and artery could be followed as far as their radial branches into the cortex. In the periosteum, two veins usually were found in conjunction with each artery. The larger periosteal vessels were arranged like the rungs of a stepladder on the posterior and lateral surfaces of the tibia, whereas on the medial surface a more irregular anastomotic arrangement was seen.

Finer Circulation of the Cortical and Cancellous Bone in the Epiphyseal and Metaphyseal Areas

The cortical bone was supplied by a frequently anastomosing network of vessels that were located in the vascular channels in the bone. The walls of the majority of these vessels were thin, containing only endothelial cells. In the cancellous bone there was a rich anastomosis between the epiphyseal-metaphyseal systems and the nutrient systems with, as a rule, one small arteriole traveling along each trabecular space. Because of the thinness of their walls, vessels could be identified with certainty only by the presence of injection media in their lumina. The method used for the examination of our tissue sections did not offer an answer to the question whether an open or incomplete endothelial lining was present in the capillary bed in the medullary spaces. Pease reported studies with the electron microscope that indicated that the endothelial lining of the vascular channels of the hemopoietic marrow is incomplete.

In general, our observations revealed that the main intra-osseous arteries were associated with companion veins, that the smallest veins progressively united and eventually left the cancellous bone of the medullary space by a route almost identical with that taken by the arteries on their way into the medulla.

COMMENT

In comparing our data with those reported in the literature, we found that our description of the epiphyseal circulation is consistent with that of other workers [2,3,15] in so far as the arterial supply is concerned. However, we disagree with several investigators [16,18,25] who have stated that the venous drainage in this region occurs through vessels leaving the bone by foramina separate from those by which the arteries enter it. Moreover, some of these workers [16,18] also have maintained that veins are more numerous than the arteries. We found that the vessels, which form the spokes in the wheellike arrangement of vessels in the epiphyseal-metaphyseal vascular system, consist of several arteries and veins in equal number. With regard to the pattern of the nutrient artery and its branches, we also agree in the main with reports of other authors [2,3,15], but in the venous system we did not demonstrate a central venous sinus with an independent channel for its outflow as described by some workers [16,18,25]. On the contrary, we found that as a general rule the veins accompanied the arteries in the human tibia. As we intimated previously, the greatest variation was found in the periosteal circulation. We agree with several recent studies [23,15] that indicate that the periosteal vessels are of little significance in supplying arterial blood to the normal tibia. This is in contrast to the classic view of the blood supply of tubular bones. We concede, however, that periosteal vessels are important as a source of capillaries for the formation of new Haversian systems and also as a potential source for the formation of new vessels in the healing of fractures.

SUMMARY

The study of fourteen limbs of adult human beings by various techniques yielded the following description of the tibial blood supply. The epiphyseal-metaphyseal regions are generously supplied by vessels entering from the pe-

riphery. These vessels anastomose with vessels from the diaphyseal area. The nutrient artery is the main source of blood to the diaphyses. It gives off ascending branches immediately after it enters the medullary cavity, and these branches disperse widely. The descending branch, on the other hand, remains as a single major vessel for some distance before finally dividing. The ascending and descending branches of the nutrient artery give off radial twigs that enter the cortex. These small arteries supply the vessels of the Haversian systems. The periosteum has a copious vascular bed that contributes only infrequent capillaries to the vascular system of the cortex. Some of these capillaries traverse the cortex from periosteum to endosteum and probably represent anastomotic links with the branches of the nutrient artery. An additional anastomosis consists of the vessels in the secondary Haversian canals, as described by Ham. The venous drainage of the diaphysis is largely toward the endosteal surface through veins that accompany the arteries. At irregular intervals, however, venous channels also drain to the periosteal surface. The venous drainage of the epiphyseal region is by way of vessels adjoining the radially arranged arteries.

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DISCUSSION

Apical Lung Carcinoma

(Continued from page 608)

Dr. George Hammond, Boston, Massachusetts: The authors point out the frequency of shoulder, arm, or neck pain as an early symptom of apical lung carcinoma. The importance of early recognition of this tumor cannot be overemphasized if we are to improve its present poor prognosis. It is admittedly a difficult diagnosis to make in the incipient period because of the mild symptoms and rather meager roentgenographic findings. The authors correctly direct our attention to the fact that the Pancoast syndrome represents the late stage of apical lung carcinoma and that the all-important early symptom is only a vague discomfort in the area of the shoulder, neck, or arm. By failing to recognize the underlying cause of this pain, physicians may delay institution of proper therapy until the disease is inoperable.

To improve our ability to make an early diagnosis, it is recommended that every patient with pain in the shoulder area have a roentgenogram made that is sufficiently large to include the shoulder, upper portion of the chest, and mediastinum. Our own roentgenologists at the Lahey Clinic recommend an anteroposterior view of this joint on a ten by twelve film placed with its longest dimension transversely. Such a roentgenogram will show not only the shoulder and lung apex but also the upper mediastinum and cervicothoracic region of the spine.

Although this paper presents nothing new, it is timely because of the increasing incidence of carcinoma of the lung. The orthopaedist should insist that the apex of the lung be included when roentgenograms of the shoulder are made, and then should train himself to look at the lung as well as the shoulder. Apical lesions have often been missed because we concentrate on the bone and fail to look at the soft tissue. This paper should stimulate us to be more alert to the possibility of apical lung carcinoma in the patient who has neck, shoulder, or arm pain. It should also stimulate us to advise even more detailed studies of this area if the patient has not responded properly to the treatment given for the disease originally diagnosed.

Dr. Dee (closing): If we are going to do anything about malignant growths we have to make a start and this offers us one place to make one. Some of these tumors are resectable, if they are caught early. We have some patients who are still alive with adenocarcinoma of the apices. But more important there are many other conditions that do not carry such a prognosis, which we, as orthopaedic surgeons, may frequently see.

As Dr. Hammond pointed out, this paper, of course, does not present anything new. It was not our purpose to present any controversial technique or syndrome, but merely to point out a pitfall in orthopaedics into which we too have fallen because in the everyday routine of the office we all see so many garden-variety run of shoulders that after a time it becomes quite monotonous.

Flexor-Tendon Ruptures in the Forearm and Hand*

BY JOSEPH H. BOYES, M.D., AND JAMES N. WILSON, M.D., LOS ANGELES, CALIFORNIA, AND JAMES W. SMITH, M.D., NEW YORK, N. Y.

Subcutaneous rupture of flexor tendons occurs infrequently, but prompt recognition and treatment can minimize disability. Little has been written about these ruptures and knowledge of the subject is limited. The object of this study is to define more clearly the incidence, etiology, mechanisms of injury, expected sites of rupture, and suggested treatment of this disorder. Eighty flexor-tendon ruptures examined from 1946 to 1959 have been analyzed for this purpose. During the same period, more than three times as many extensor-tendon ruptures were recorded.

Von Zander's report in 1891 of a rupture involving the flexor pollicis longus tendon was the first recorded flexor-tendon rupture in the forearm or hand. Mason, in 1930, considered flexor-tendon ruptures to be rare. He reported two cases of subcutaneous rupture in normal flexor tendons, and found only two others in the literature. He included, from Kanavel's files, ten cases of actual or impending flexor-tendon rupture associated with tuberculous tenosynovitis. Haldeman and Soto-Hall, reviewing 100 ruptures of muscles and tendons in all sites, found flexor-tendon ruptures of the forearm and hand to represent only 3 per cent of the total. Bunnell listed twenty cases, sixteen of which were from his own records, of flexor-tendon ruptures, which occurred at various sites.

The definition of spontaneous rupture is not clear and we have chosen to avoid this term. Its use should be restricted to those ruptures that occur within the tendon substance. It should not be used if the ruptures are directly related to underlying or associated pathological changes. Only three of our cases could be classified in this category.

Normal tendon seldom ruptures because it is the strongest link in the musculotendinous chain[8]. The insertion, musculotendinous junction, muscle substance, or even the muscle origin will almost invariably give way before the tendon itself yields. Experimental studies by McMaster[23], and more recently by Fink and Wyss, lend support to this view. Stucke found that loads of up to about 400 kilograms were required before the Achilles tendon of young people ruptured. Clinical confirmation of tendon strength is found in Adams' report of a seaman who caught the long and ring fingers of the right hand in a door, amputating the distal phalanx of the long finger. As he withdrew his hand from the closed door, the tendon of the flexor digitorum profundus, along with some muscle, was pulled away with the amputated part. Last published two cases from the Hunterian Collection in which similar separations occurred at the musculotendinous junction.

The intrinsic strength of tendon is further verified by showing experimentally that at least half of the fibers require division before tendon rupture can be produced, even under extreme stress[22]. This finding suggests that when ruptures occur within the tendon, the tensile strength has been reduced at least 50 per cent prior to the application of the actual distracting force. This weakening can result from direct injury, indirect injury, or disease. Direct injuries occur with lacerations or crushing trauma and can be followed by immediate or delayed rupture. Experimentally, after segmental injury, delayed rather than immediate

* Read at the Annual Meeting of the American Society for Surgery of the Hand, Chicago, Illinois, January 23, 1960.

ruptures are more likely to follow [23]. Indirect tendon injuries may result from forces acting in direct opposition to the muscular force. The magnitude of such forces has an important bearing on the degree of injury. The status of muscle contraction at the time of injury may be significant since most ruptures are reported to occur when a muscle is in its maximum contracted state [23, 28]. Flexor-tendon ruptures, then, should occur most frequently after sudden, forceful extension of a flexed finger. Stucke believed that indirect violence alone is incapable of rupturing a healthy tendon, and suggested that attrition of the tendon must have preceded it when rupture occurs within the tendon substance.

Disease may affect the tendon. Historically, obesity, gout, syphilis, gonorrhea, tuberculosis, typhus, typhoid, and pyogenic infection have all been cited as etiological factors which may be associated with tendon disruption [24]. Current medical literature, however, suggests that they play an infrequent role.

More recently, bone abnormalities [10, 18], rheumatoid arthritis [31], and tenosynovitis [29] have been cited with increasing frequency as etiological factors [19]. Although tumor is often listed as a causative agent, there are no reported cases in which it was proved directly responsible [26].

Toygar suggested that in some cases a genetic factor may account for the diminished tensile strength that precedes rupture, but there is little evidence to substantiate such a theory.

Senility has often been considered responsible for the degenerative changes seen on microscopic examination of a ruptured tendon [14]. However, because these tendon ruptures occur so frequently in young, healthy adults [3, 4], factors other than aging must play a significant role. Håstad, Larsson, and Lindholm, using radioactive sodium to study the blood flow in Achilles tendons, demonstrated a decreasing vascularity after the third decade of life. Because of the frequency of ruptures before the fourth decade and because the precipitating force is often relatively mild, they concluded that probably neither the effects of aging nor the vascular changes are major factors. The observations of Maydl, Albrecht, Pirker, and Arner and Lindholm, that ruptures are common in individuals who have participated intensively in sports or physical activities suggest that ruptures may be related to muscle hypertrophy.

Occupation has long been recognized as a significant factor in tendon ruptures. Extensive use of a specific tendon may result in chronic synovitis, attrition, and eventual rupture [19]. This sequence of events has been demonstrated in ruptures of the extensor pollicis longus [17, 26]. With repeated and prolonged muscle stimulation, Borsay and his associates were able to produce similar degenerative changes and eventual tendon rupture in hares and dogs.

PRESENT INVESTIGATION

In this paper we have studied eighty subcutaneous ruptures of flexor tendons in seventy-eight patients examined over a thirteen-year period. The involved tendons included the flexor digitorum profundus, flexor digitorum sublimis, and flexor pollicis longus. In some instances, multiple flexor-tendon ruptures were present.

The average age of these seventy-eight patients was 40.9 years (men 39 years and women 42.9 years). The youngest patient was sixteen years of age and the oldest sixty-one. There were sixty men and eighteen women. The right hand was involved in forty-three patients and the left in thirty-five. In forty-five instances the dominant hand was injured and in thirty-two, the subordinate.

Rupture of one or both flexor tendons of the ring finger was noted twenty-three times, the long finger nineteen times, the thumb fifteen times, the little finger twelve times, and the index finger eight times. In one instance all flexor profundus tendons were ruptured. In thirty-six instances the rupture occurred in persons who might be classed as non-manual workers, whereas in forty-two cases the patient was doing manual labor. Ruptures of the flexor profundus tendon were

noted in thirty-nine patients. The sublimis tendon was ruptured in eight patients, and in seventeen patients both profundus and sublimis were involved. There were fifteen ruptures of the flexor pollicis longus tendon.

The tendons of the flexor digitorum profundus were most frequently ruptured in this series (Table I). The forty ruptures of only the profundus tendons in thirty-nine patients represented 50 per cent of the total number of flexor-tendon ruptures. By contrast, ruptures of the sublimis tendon alone represented only 10 per cent of the total. Combined ruptures (flexor profundus and sublimis) and ruptures of the flexor pollicis longus were of almost equal frequency, each representing about one-fifth of the total.

TABLE I

DISTRIBUTION OF EIGHTY RUPTURES

Tendon Involved	No. of Patients	No. of Ruptures	Percentage of Total No. of Ruptures
Profundus	39	40	50
Sublimis	8	8	10
Profundus and sublimis	17	17	21
Flexor pollicis longus	14	15	19
Totals	78	80	

TABLE II

DISTRIBUTION OF TENDON RUPTURES ACCORDING TO AGE AND SEX
SEVENTY-EIGHT PATIENTS

Tendon Involved	No. of Ruptures in Male Patients	No. of Ruptures in Female Patients	Ratio: Male to Female		Average Age in Years: Male Patients	Female Patients	Both Groups
Profundus	27	12	2.3:1		32.6	36.5	34.6
Sublimis	7	1	7:1		37.9	30	33.9
Profundus and sublimis	14	3	4.6:1		42.4	51	46.7
Flexor pollicis longus	12	2	6:1		43.2	54	48.6
Totals	60 (77 per cent)	18 (23 per cent)	3.3:1	Total Group	39	42.9	40.9

Flexor-tendon ruptures occur predominantly in male patients (Table II). Although the degenerative changes that accompany tendon aging are often considered to play a role in the predisposition of tendons to rupture, this was not substantiated in our study, the average age of all patients being 40.9 years (Table II). There was an unexplained difference between the average age of those patients in the profundus tendon group and those in the combined flexor sublimis and profundus group and the flexor pollicis longus group. The higher average age of the five females with both combined ruptures and with ruptures of the flexor pollicis longus deserves comment. The average ages in these two small groups were 51.0 years and 54.0 years, respectively—both considerably higher average ages than in any of the other groups. Three of these five patients had chronic rheumatoid arthritis, which ultimately played a role in the ruptures.

In Table III it is shown that 8.8 per cent of the flexor-tendon ruptures occurred in persons under twenty-one years of age. The group is included to make 27.8 per cent in those under thirty-one years of age, and 83.4 per cent in those under fifty-one years. Only 2.5 per cent of the ruptures in this series occurred

TABLE III

DISTRIBUTION OF SEVENTY-NINE RUPTURES ACCORDING TO AGE OF PATIENT, IN DECADES

Tendon Ruptured	11 to 20 Years	21 to 30 Years	31 to 40 Years	41 to 50 Years	51 to 60 Years	61 to 70 Years
Profundus	4	12	12	9	3	
Sublimis		2	4	2		
Profundus and sublimis	1	1	3	8	3	1
Flexor pollicis longus *	2		3	3	5	1
Totals	7 (8.8 per cent)	15 (19 per cent)	22 (27.8 per cent)	22 (27.8 per cent)	11 (13.9 per cent)	2 (2.5 per cent)

* No age recorded in one patient.

TABLE IV

LOCATION OF INVOLVEMENT IN SEVENTY-SEVEN HANDS

Tendon Ruptured	Right Hand	Left Hand	Dominant Hand	Subordinate Hand
Profundus *	19	19	21	17
Sublimis	7	1	6	2
Profundus and sublimis	9	8	10	7
Flexor pollicis longus	8	6	8	6
Totals	43	34	45	32

* Two cases not recorded.

TABLE V

DISTRIBUTION OF INVOLVEMENT IN SEVENTY-SEVEN DIGITS

Tendon Ruptured	Thumb	Index Finger	Long Finger	Ring Finger	Little Finger
Profundus *		1	12	18	6
Sublimis		1	3	3	1
Profundus and sublimis		6	4	2	5
Flexor pollicis longus	15				
Totals	15	8	19	23	12

* One case, all flexor profundi ruptured; one case involved digit not recorded.

in people over sixty years of age, whereas 55.6 per cent were present in individuals between thirty-one and fifty.

Because the degenerative changes produced by excessive use appear to play a significant role in ruptures of the extensor pollicis longus tendon, this relationship was examined in our series. The dominant hand is used more frequently than the subordinate hand and is usually preferred when maximum strength and effort are required. Although the incidence of rupture was greater in the dominant hand than in the subordinate, the difference was neither striking nor statistically significant (Table IV). There also seemed to be little difference in the incidence of rupture between the left and right hands (Table IV).

Table V shows that ruptures in the ring and long fingers were more frequent than in the index and little fingers, not only in profundus and sublimis injuries, but in the over-all figures as well.

A study of the occupation and the type of work indicates that these are

TABLE VI

TYPE OF EMPLOYMENT—SEVENTY-EIGHT PATIENTS

Tendon Ruptured	Manual Laborers	Non-manual Workers
Profundus	20	19
Sublimis	5	3
Profundus and sublimis	10	7
Flexor pollicis longus	7	7
Totals	42	36

TABLE VII

ETIOLOGICAL FACTORS—EIGHTY RUPTURES*

Tendon Ruptured	Indirect Injury		Direct Injury
	Hyper-extension	Flexion against Force	Crush or Laceration
Profundus	19	5	12
Sublimis	5	1	2
Profundus and sublimis	5	3	4
Flexor pollicis longus	4	2	2
Totals	33 (41 per cent)	11 (14 per cent)	20 (25 per cent)

* Disease was responsible for sixteen (20 per cent) of the eighty ruptures.

TABLE VIII

RELATION OF FORTY-NINE INSERTION RUPTURES TO ETIOLOGICAL FACTORS

Tendon Ruptured	Hyperextension	Forced Flexion	Crush or Laceration	Disease
Profundus	18	4	6	1
Sublimis	3		2	
Profundus and sublimis	4	1	2	1
Flexor pollicis longus	4		2	1
Totals	29 (59.2 per cent)	5 (10.2 per cent)	12 (24.5 per cent)	3 (6.1 per cent)

unrelated to either the incidence of rupture or to the type of rupture (Table VI). However, the type of injury and the presence of associated pathological changes do seem to be closely related to the incidence of rupture (Table VII), as well as to the site of rupture (Table IX).

Among the eighty ruptures in this series indirect trauma (Table VII) accounted for forty-four, whereas direct trauma was related to rupture in twenty. In the sixteen remaining cases no history of trauma could be obtained from the patients to explain the rupture. However, in all but three of these sixteen tendons, the presence of some underlying disease readily explained the reason for rupture. Frequently, several factors were involved in the rupture. Twenty per cent of the eighty tendons had pathological changes or disease that could explain the rupture.

Sudden hyperextension of the involved finger was considered responsible for rupture in 41 per cent of the involved tendons, whereas in 14 per cent, rupture occurred when the patients were attempting to flex against resistance. These ruptures appeared related to the physical state of muscle. Since the elasticity coefficient of muscle decreases with contraction and shortening, the greater the

TABLE IX

SITE OF SEVENTY-EIGHT RUPTURES

Tendon Ruptured	Insertion	Mid-segment of Digit	Palm at Lumbrical Origin	Carpal Tunnel	Wrist	Musculotendinous Junction
Profundus	29	6		3		
Sublimis	5			1		2
Profundus and sublimis	8	2	3	4		
Flexor pollicis longus	7	1		2	3	2
Totals	49 (62.8 per cent)	9 (11.5 per cent)	3 (3.9 per cent)	10 (12.8 per cent)	3 (3.9 per cent)	4 (5.1 per cent)

TABLE X

OPERATIVE PROCEDURES—EIGHTY TENDONS

Tendon Ruptured	Operation Advised (No. of Tendons)	Operation Not Advised (No. of Tendons)
Profundus	15	25
Sublimis	1	7
Profundus and sublimis	17	0
Flexor pollicis longus	14	1*
Totals	47	33

* Rheumatoid activity precluded surgery.

degree of muscle contraction, the greater will be the danger of rupture as a result of indirect violence [21, 28].

Absence of the neuromuscular coordination normally present during planned movements may account for the frequency of rupture associated with sudden unopposed digital hyperextension [6]. Normal coordination allows for the gradual relaxation of one group of muscles as the opposing group contracts. This automatic adjustment of muscle tension and length aids in resisting sudden forced movement. Sudden unexpected hyperextension may initiate a strong reflex flexion action, and rupture may result.

In the twenty ruptures following direct injury, crushing preceded the rupture in fourteen, whereas partial laceration preceded rupture in six. All except two of the crushing injuries had an immediate rupture; the two latent ruptures occurred at three weeks and two months, respectively. In those instances where partial division must have occurred at the time of initial injury, latent ruptures were noted as early as two weeks and as late as four months after injury.

The sites of rupture are recorded in Table IX. Ruptures at the tendon insertion accounted for 62.8 per cent of seventy-eight ruptures, and ruptures at the musculotendinous junction for 5.1 per cent. No ruptures were known to occur within the muscle substance or to involve the muscle origin. Twenty-nine (87.8 per cent) of the thirty-three patients having hyperextension injury had their ruptures at the tendon insertion. Twenty-nine (59.2 per cent) of the forty-nine ruptures at the tendon insertion were produced by hyperextension forces (Table VIII).

Twenty-five ruptures occurred within the tendons (Table IX). In all except three, the rupture was associated with pathological changes. There were nine tendon ruptures elsewhere within the fingers than at the insertion; direct trauma had occurred in all except one of these. In the other sixteen ruptures, the tendons

ruptured in the palm, carpal tunnel, or wrist. Eight of these sixteen ruptures were associated with rheumatoid arthritis; in two there were congenital abnormalities of the carpal bones; two followed fracture of the radius; and one was associated with a non-specific tenosynovitis. Rheumatoid arthritis was associated with six of the ten ruptures in the carpal tunnel, and an anomaly of a carpal bone in three. These anomalies of the carpal bones accounted for three ruptures in two patients. A roughened hook of the hamate was thought responsible for a combined rupture of both flexor tendons of the little finger and the profundus tendon of the ring finger. The capitate was bipartite in another patient in whom both the profundus and the sublimis tendons to an index finger had ruptured at this level.

There were only three patients with true ruptures of apparently normal tendons; one had a rupture of the flexor pollicis longus tendon and two had ruptures of both the profundus and the sublimis tendons. The rupture of the flexor pollicis longus had occurred at the metacarpophalangeal joint in the dominant hand of a radio engineer, thirty-two years old, who was in excellent health and who was injured while pinching a battery clamp. Neither history nor operative findings explained why the tendinous portion of the flexor pollicis longus had ruptured.

The other two patients had ruptures of both the sublimis and the profundus tendons at the level of the lumbrical origin. One, a truck driver, thirty-seven years old, ruptured these tendons in the long finger of his right, dominant hand as he was pulling a small cart. The other, an aircraft inspector, forty-two years old, noted sudden hyperextension of the little finger of his right, dominant hand as he pushed open a light overhead cockpit door. The operative findings failed to explain the ruptures in either case.

Table IX emphasizes that in twenty-five of forty ruptures of the flexor profundus tendon the patient was advised against operation. In fifteen digits, some type of operative procedure was carried out. This was usually tenodesis of the distal interphalangeal joint to stabilize the finger tip in slight flexion. A persistently painful proximal tendon remnant in the palm was removed, but tenderness of this origin usually subsided without treatment. In two instances the ruptured profundus tendon became adherent at the level of the bifurcation of the sublimis tendon and limited function of the latter so that preoperatively both tendons were thought to be ruptured. In these digits, removal of the profundus freed the sublimis and improved function of the middle joint. In three cases, the patient was seen shortly after rupture at the insertion of the tendon, and delayed repair was done. In only one patient was a tendon graft performed. In this instance, it was found at operation that the associated damage to the sublimis necessitated removal of both tendons.

Loss of flexion of the distal joint, especially in the ring and little fingers, may prove to be only a minor disability. A careful appraisal of the patient's subjective complaints frequently will lead to the decision that no treatment is indicated. It is our opinion that a distal joint fixed in the position of function by tenodesis or athrodesis is the treatment of choice for the average patient. The difficulties and complications which can follow an unsuccessful attempt to restore flexion of the distal joint by profundus tendon-grafting through an intact sublimis are well known, and the risk of increasing the disability far outweighs the possible improvement to be gained from a graft.

In isolated ruptures of the sublimis tendon function of the finger was seldom impaired, and complaints were minimum in this group. In two patients, limitation of extension of the middle joint was improved by the use of a safety-pin splint. In one instance where rupture occurred at the musculotendinous junction, there was an associated tenosynovitis which responded favorably to immobilization. In only one patient was surgery required because the injury had produced a severe flexion deformity of the middle joint. Excision of the distal sublimis remnant in

this instance improved extension of the middle joint from 96 to 165 degrees. No significant disability was present in any of the patients who were not operated on.

In all combined ruptures, an operative procedure was advised. In one finger amputation was necessary for persistent joint stiffness, which followed an open dislocation of the middle joint. In ten ruptures the tendon remnants were excised and a graft inserted.

Eleven of the fourteen patients with ruptures of the flexor pollicis longus tendon had reconstructive procedures performed. In two instances (one a disruption at the insertion of the tendon and the other a latent rupture at the distal flexion crease) it was possible to advance the tendon to the old insertion. In eight patients the remnants of the old flexor pollicis longus tendon were excised and a graft inserted. In one patient the sublimis tendon of the ring finger was transferred to the flexor pollicis tendon. In another patient active rheumatoid arthritis made surgery impossible. In two patients tendon grafts that extended from the insertion to the level of the wrist were required; in the remainder the proximal suture line was placed in the thenar eminence. We prefer this level for suture, if possible, since it minimizes the hazard of median neuritis, which can occur after full-length grafts.

DISCUSSION

This study indicates that ruptures of the flexor tendons are not rare. Probably, many ruptures have not been reported. After isolated rupture of the profundus or sublimis, many of our patients had minimum disability. Had there not been disability benefits involved, or a company requirement that all injured personnel be seen by a physician, a significant number of these persons might not have sought medical care. A few patients had undergone extensive examinations for rare and exotic muscular and nervous disorders before the diagnosis of tendon rupture was considered.

We have attempted to emphasize that no treatment is frequently the best treatment for ruptures of the profundus and sublimis tendons. However, in some cases a simple surgical procedure such as tenodesis or excision of a portion of the ruptured tendon will be required. It is only when flexor function has been severely compromised or lost that radical surgery is essential.

In our experience, flexor-tendon ruptures appear most frequently in healthy men between the ages of thirty and fifty years. Occupation seems unrelated to ruptures.

Rupture usually follows a definite episode of either direct or indirect trauma. It is often associated with a snap or pop at the site of separation, but there may be little or no associated pain. Statistically, tendons of the ring and long fingers will be ruptured most frequently.

The direct or indirect traumata that precede rupture are usually crush injuries, lacerations, sudden and unexpected digital hyperextensions, and forced flexions against resistance. In normal tendons these ruptures will occur at either the tendon insertion or the musculotendinous junction. Where tendons have been weakened by trauma or disease, rupture may occur within the tendon substance. In these latter ruptures, a history of rheumatoid arthritis, tenosynovitis, or wrist fracture is common. It is these pathological processes that are often responsible for the weakening of the tendon that precedes rupture.

SUMMARY

Eighty ruptures of flexor tendons, occurring over a thirteen-year period in the fingers in seventy-eight patients, were studied. The tendons involved were the flexor digitorum profundus, flexor digitorum sublimis, and flexor pollicis longus.

The majority of ruptures occurred in young patients, suggesting that degenerative changes accompanying aging were not directly responsible for rupture.

The force producing the rupture was often slight, indicating that some underlying factors had already predisposed the tendon to rupture.

Flexor-tendon ruptures were found to occur most frequently after crushing injuries, lacerations, hyperextension injuries, and as the result of forced flexion against resistance. In 67.9 per cent of the tendons, these ruptures were found at the insertion or musculotendinous junction. In 31.9 per cent, they occurred within the tendon substance.

Hyperextension injuries most often produced ruptures at the insertion. Lacerations and crushing injuries were also frequently followed by rupture of a flexor tendon at its insertion or elsewhere within the finger. Ruptures resulting from forced flexion against resistance produced injuries at the tendon insertion, except in those instances where the tensile strength of the tendon had been decreased by pathological changes. In 20 per cent of all the ruptures, pathological changes in the tendon accounted for rupture and in nearly all of the tendons with these changes the ruptures occurred in the palm, carpal tunnel, or wrist.

It is emphasized that surgical treatment is often unnecessary. Surgery is essential only when flexor function has been severely compromised or lost.

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Primary Repair of Flexor Tendons*

BY CLAUDE E. VERDAN, M.D., LAUSANNE, SWITZERLAND

Among some of the difficult problems still to be solved in hand surgery, repair of flexor tendons in the digital sheath is not the least.

It is generally agreed that in a single fresh cut of the flexor profundus between the distal insertion and the middle third of the middle phalanx, primary suture can give good results (Figs. 1-A through 2-C). It is quite different, however, when the wound is located more proximally and both tendons are cut. Most authors say it is wise not to undertake immediate tendon repair when both flexor tendons are divided in Bunnell's no man's land. Treatment is limited to closing

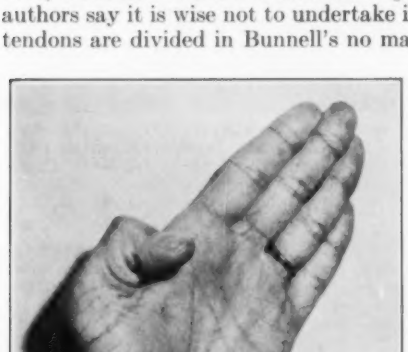

FIG. 1-A

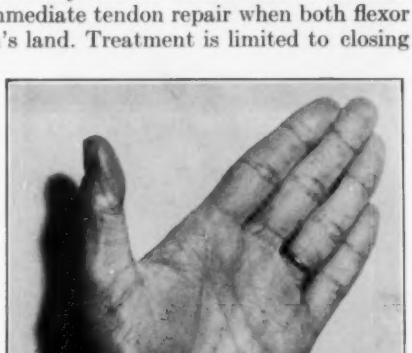

FIG. 1-B

Result of primary suture of the flexor pollicis longus, severed in the interphalangeal fold.

the wound, preceded by economical excision of the wound edges, if they are contused. Secondary tendon repair is postponed for two or three weeks. After this interval, excision of the sublimis is recommended, followed by a long tendon graft of the profundus, extending from the distal attachment of this tendon up to the proximal half of the palm (Figs. 3-A and 3-B).

According to my own experience, this operation is not only one of the most difficult undertakings in reparative surgery but also one which gives the most uncertain results. It is known that these results depend on the selection of cases for the operation. A badly performed or unsuccessful primary repair can, of course, jeopardize the success of a secondary graft; but it must also be recalled that division of the two neurovascular bundles of a finger will cause trophic disorders, and these make secondary repair very difficult.

Skill, patience, and painstaking attention to details are required of the surgeon.

The good results obtained by Boyes[1] and Pulvertaft[8] (70 to 80 per cent success) are not always attained. I, personally, during the past seven years have performed about sixty tendon-grafting procedures. I must admit, however, that my percentage of good reults is not so high.

Without giving details concerning the technical difficulties that may impair the final result, I would say that, at present, a tendon graft is still the exclusive job of the specialist or, at least, the specially trained surgeon. Usually these

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FIG. 2-A

FIG. 2-B

FIG. 2-C

Figs. 2-A, 2-B, and 2-C: The flexor profundus alone was severed in the middle third of the middle phalanx of the index finger. Only primary tendon repair was performed (no tenolysis).

Figs. 3-A and 3-B: Cut of both flexor tendons in no man's land of the little finger. Photographs show the result five months after tendon-grafting.

FIG. 3-A

FIG. 3-B

FIG. 4-A

FIG. 4-B

FIG. 4-C

Figs. 4-A, 4-B, and 4-C: Cut of both flexor tendons in the index finger, of the flexor profundus and one-half of the sublimis in the long finger, and of one-half the profundus in the ring finger. Only primary repair was performed; the patient did not wish a secondary tenolysis.

surgeons are not the ones who see the patients in the emergency room. It is, most frequently, the general practitioner or the intern. Under these circumstances, it is justified and wise not to perform primary repair.

However, I would like to show that the surgeon who is trained to perform a successful graft of the flexor tendons is also capable of performing a successful primary tendon repair—if the wound is fresh and cleanly cut—without crushing the tissues, if he applies some sound technical rules.

In my book *Chirurgie Réparatrice et Fonctionnelle des Tendons de la Main*, which I wrote in 1951, I came to the conclusion, after giving all the reasons for and against it, that primary repair of the flexor tendons is not only advisable but also possible, not only in the areas known to be favorable for primary repair, but also in the osteofibrous tunnel of the finger. However, this primary repair must solve the mechanical and physiological problem of gliding.

I described in my book a new technique I applied which utilizes stainless-steel transfixion pins to hold the ends of the lacerated tendon in contact with one another in a part of the sheath that is intact, without placing any sutures in the tendon ends.

Although some good results have been obtained with this method (Figs. 4-A, 4-B, and 4-C), I have now decided that this is not the best possible solution to the difficult problem which Bunnell, Mason and Allen, Koch, and others have tried to solve.

I have now modified this first technique and have used systematically a second method of blocked sutures, which includes resection of the sheath and greater precision in the alignment of the cut ends.

Actually, in the first method, the ends of the flexor profundus were sometimes too loose inside the sheath from which the sublimis had been removed. Within the intact sheath the tendon ends could have some play and thus, during healing, a brush of regenerating "unsatisfied" fibers was formed. Since the sheath

participates in the healing of the tendon, the healed tendon did not glide as well as one might hope for.

Therefore, I now resect about one inch of the sheath at the site of the suture, as Mason did, and suture the ends of the tendon together very accurately with four small epitendinous stitches at the four cardinal points, using fine 000000 arterial silk, as is done for a nerve suture.

This accurate but fragile repair will not resist the traction of the muscles. However, protection is achieved by immobilizing the tendon with two stainless-steel pins, a procedure which is much easier than Bunnell's "suture at a distance", using a pull-out wire.

The pins that pass through the skin, the sheath, and the tendon are placed transversely; one distal and one proximal to the suture.

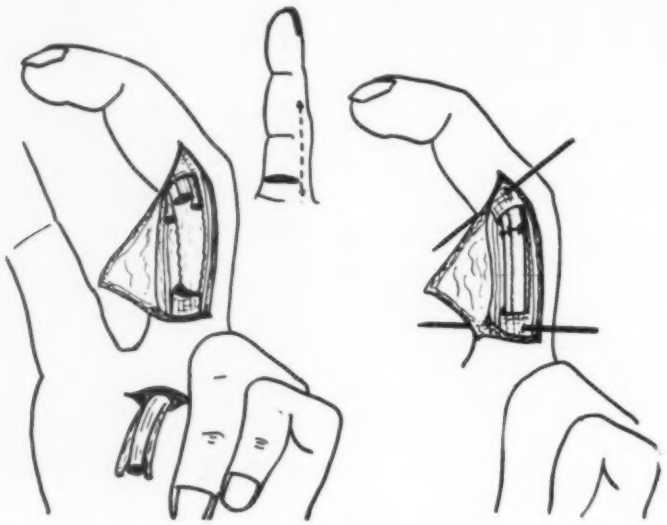

FIG. 5

Technique of primary blocked suture of the flexor profundus when both flexor tendons are severed in no man's land; resection of the flexor sublimis and the digital sheath.

TECHNIQUE

After economical excision of the margins of the transverse wound caused by the accident, the incision for the repair of the tendon is made, using a classic mid-lateral opening, without much regard for the wound produced by the trauma. The approach to the sheath goes dorsal to the neurovascular pedicle; the nerve, if severed, is repaired only at the end of the operation.

First the sublimis is resected. For this procedure, the lesion of the tendon sheath caused by the accident generally is too small. This opening is therefore enlarged and used for the extraction and resection of the two small distal slips.

The proximal end of the flexor sublimis tendon, which has retracted into the palm, is located through a short transverse incision in the distal palmar crease. It is divided as far proximally as possible. Only the profundus is replaced in the sheath and pushed into the digital canal.

At this time it is important to place the finger in the proper semiflexed position in which it will be immobilized and in which the pins will be inserted before the tendon is sutured. In doing this one must look for the "position of rest", which changes with flexion or extension of the wrist. With the finger in this position the lacerated ends of the tendon will probably have no relation to the acci-

FIG. 6-A

FIG. 6-B

Figs. 6-A, 6-B, and 6-C: Both flexor tendons were severed in no man's land of the long finger. In the index finger, there was a cut of the flexor profundus between the proximal and middle thirds of the middle phalanx. Figs. 6-B and 6-C show the result seven months after primary repair.

Figs. 7-A and 7-B: Both flexor tendons were severed in the middle third of the proximal phalanx of the ring finger. The photographs show the result after primary suture of the profundus and one collateral nerve.

FIG. 6-C

FIG. 7-A

FIG. 7-B

dental opening in the sheath. Consequently, this opening in the sheath is disregarded from this point on.

Over the juncture of the two ends of the tendon, the sheath is opened or the existing opening is enlarged so that the sheath is excised over a distance of about one inch. This is done in such a way that the future scar at the site of tendon repair will slide freely in both directions without contacting the sheath. The amount of

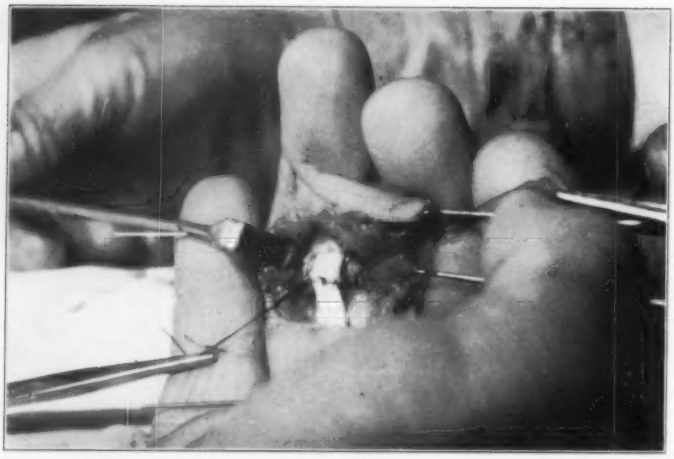

FIG. 8-A

FIG. 8-B

FIG. 8-C

Figs. 8-A, 8-B, and 8-C: Cut of both flexors in the distal third of the first phalanx of the ring finger. Primary blocked suture of the flexor profundus was performed. Figs. 8-B and 8-C show the functional result at follow-up two years after operation.

sheath resected is determined by estimating the physiological gliding amplitude at this level (Fig. 5).

This means that most frequently the suture line will not be at the same location as the original lesion. If the finger was extended at the time of injury—a very rare occurrence—the suture line will be proximal to the site of injury. If, on the contrary, the finger was flexed at the time of injury—which quite frequently happens because of an instinctive protective reflex which comes into play at the time of injury—the suture line will lie near the distal end of the middle phalanx. This is very important because if the tendon scar does become fixed to the middle phalanx, the point of attachment will be so far distal that the tendon will preserve enough excursion within the finger to produce flexion of the proximal interphalangeal joint—a motion of capital importance for function of the digit.

Furthermore, by excising the sheath in an intact zone, the tendon scar will be surrounded by subcutaneous fat and not by a fibrous tunnel of fixed diam-

FIG. 9-A

FIG. 9-B

FIG. 9-C

Figs. 9-A, 9-B, and 9-C: Tenolysis. Cut of both flexors at the level of the proximal interphalangeal joint of the index finger. Because the primary repair failed, tenolysis was performed two and one-half months later.

eter. The remainder of this sheath suffices to prevent dislocation of the tendon, just as it does in the technique of tendon-grafting, in which resection of the sheath is still more extensive.

The finger is immobilized in a resting position by a padded dorsal splint. If the flexor tendon of the thumb is being repaired a cast should be used.

I have not yet ventured to use corticoids after operation.

After a maximum of three weeks, the splint and the blocking pins are removed. Systematic active exercises, including pushing on the palmar side of the phalanx, are then started cautiously and increased gradually to mobilize the tendon. Sometimes, gentle passive mobilization during the fourth week after operation will rupture fresh adhesions, and this may improve the end result considerably.

Eight to ten days after the beginning of mobilization of the digit the patient is permitted to resume partially his former activities.

Thus, the time of immobilization and the period of early rehabilitation amount to from four to five weeks of disability (Figs. 6-A through 8-C).

Tenolysis after Unsuccessful Primary Suture

It will be necessary to perform a secondary tenolysis: (1) if after three months there has been no functional improvement and the active motion remains much less than the passive movement in the joints of the involved finger; (2) if the patient thinks the result is not sufficient for the needs of his professional task; or (3) if there is complete adhesion of the tendon, as occurred in 25 to 30 per cent of the patients.

This operation requires not only the freeing of adhesions but often a resection of the tendon sheath, and of the normal pulleys as well, in digits in which adhesions of the tendon cover a long distance. As in the first step of a tendon graft, only a short fibrous ring is left as a pulley on each phalanx. Removal of the sheath is continued until traction on the flexor profundus at the upper half of the palm causes complete, effortless flexion of the finger. Elastic bandages

FIG. 10-A

FIG. 10-B

FIG. 10-C

Figs. 10-A, 10-B, and 10-C: Tenolysis. Both flexor tendons of the long finger were severed at the metacarpophalangeal fold. Failure of primary suture of the profundus and resection of the sublimis was followed by complete adhesions of the tendons. Normal function was regained after tenolysis.

are applied after operation, and mobilization is begun as soon as the postoperative pain will allow.

My friend, Jacques Michon, from Nancy, and I demonstrated that secondary tenolysis, the indications of which are many, can be particularly useful after an unsuccessful primary suture as well as after an unsuccessful tendon graft. However, it is advisable to wait six months before performing a tenolysis for an unsuccessful tendon graft if one wishes to avoid spontaneous rupture of the graft. For primary repair of flexor tendons a delay of three months seems sufficient (Figs. 9-A through 10-C).

RESULTS

Lesions of the flexor tendons in no man's land are relatively rare. Although the hand surgeon is seldom called on to repair fresh lacerations, he is very often requested to perform grafts on patients sent to him from all over the country. These second operations, of course, must be performed according to the prescribed program for tendon-grafting.

Since 1952, end results after primary suture of flexor tendons in no man's land, performed according to the technique described, are available for five thumb flexor tendons and fourteen flexor tendons in the four other digits, a total of nineteen cases. The results obtained in three other patients whose digits are still under treatment are too recent to be considered.

In the thumbs the amplitude of active motion, measured at the interphalangeal joint, ranged from 45 to 80 degrees.

In the lesser digits, ten of fourteen fingers have a range of active motion between 20 and 95 degrees for the proximal interphalangeal joint, and between 10 and 30 degrees for the distal interphalangeal joint. None of these ten fingers required additional intervention.

There were three failures due to complete adhesions of the tendon and one

partial failure due to flexion contracture of the proximal interphalangeal joint; these fingers required reoperation. In one of these fingers a tendon graft was a failure. The other three were treated with tenolysis, resulting in a functional recovery which was almost normal in one and perfect in another. In the third, complete flexion was regained but a new progressive limitation of extension was produced by flexion contracture of the proximal interphalangeal joint.

I recently reoperated on this last-mentioned finger, resecting the retracted new sheath and the volar ligament of the proximal interphalangeal joint. Luckily, the final result, at the time of writing, is good, the patient having worn an elastic dorsal splint.

Generally, results, as in the case of grafts, seem to depend on the trophic state of the finger, and, therefore, on the presence of concomitant laceration of the digital nerves. Possibly, the results also depend, to some extent, on the integrity of the periosteal covering of the phalanx.

It is worthy of note that even a poor result in the index finger can give useful function since pinch between the thumb and index finger will be possible. The absence of complete flexion is a less severe handicap in the index finger than in the other three fingers, as was pointed out by Pulvertaft in connection with tendon grafts. The same argument is applicable for primary sutures.

Multiple Sections

I believe that when the flexors of several fingers have been cut simultaneously, repair by a secondary graft becomes particularly difficult. To perform multiple tendon grafts at one operation is a very long and detailed procedure.

The time required for the functional re-education will be longer, adhesions of grafts will be more difficult to liberate, and the patient's period of incapacity will be longer.

When the flexor tendons of several fingers have been severed, primary repair —a relatively short operation—seems all the more justified. In the hands of a specially trained surgeon primary repair does not endanger the future of the hand and offers the possibility of restoring adequate function by performing only a secondary localized tenolysis on one or more fingers or, if need be, a complete tenolysis. Moreover, after primary repair the possibility of doing a later graft still exists.

If the patient can start working soon after the primary suture, dystrophic changes in the injured hand will be minimized. These changes are so frequent and sometimes so serious after the flexor tendons of several fingers have been severed, that one often hesitates to undertake the risk of a secondary tendon graft. In the meantime, the tendon sheaths contract and become obliterated, the articulations become stiff, and success is jeopardized.

CONCLUSIONS

Although my series of nineteen cases of primary sutures and sixty tendon grafts is a modest one, I believe, nonetheless, that it entitles me to state that primary repair of the flexor tendons in no man's land, performed according to certain technical rules, is a valid operation for fresh, cleanly cut wounds.

Results of primary repair appear to me, as far as quality is concerned, the same as, if not superior to, those obtained by tendon grafts.

Regarding fresh simultaneous lacerations of the tendons of several digits, my preference for primary suture becomes imperative.

SUMMARY

The rule hitherto adopted that tendon grafts should be performed in preference to primary repair of tendons cut in Bunnell's no man's land is too rigorous. Delayed tendon-grafting is only justified for those physicians having no experience in hand surgery or when general or local conditions do not allow immediate repair.

For cleanly cut, fresh wounds, specialists capable of performing tendon grafts correctly, are *a fortiori* qualified to effect successful primary sutures. The technique proposed consists in excising the sublimis, suturing the profundus accurately with fine epitendinous stitches, resecting the sheath in the region of the repair, and immobilizing the ends of the tendon with two transverse stainless-steel pins to prevent tension on the delicate suture line, which is comparable to that in a nerve suture.

Should adhesions hinder gliding, a secondary tenolysis will produce functional results often better than those obtained by tendon grafts.

For multiple simultaneous flexor-tendon lacerations in several digits, primary repair is even more justified than for a tendon laceration in one digit.

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DISCUSSION

Dr. Michael L. Mason, Chicago, Illinois: The only area of the hand where there is major disagreement concerning primary tendon repair is in what has been euphemistically called no man's land, and I should like to confine my few remarks to that phase of the problem. It was not too many years ago that the grafting of tendons in this area of the hand seemed to be an insurmountable problem and was undertaken with little assurance of success. In fact, the solution for the problem of a motionless finger was often amputation. Even today, tendon-grafting in the digits is undertaken only when conditions are satisfactory and by surgeons well trained in the technique. Even so, tendon grafts in the digits are by no means 100 per cent successful and many ingenious measurements are made to gauge the degree of return of function. No one suggests, however, that tendon-grafting should be abandoned; but, simply, that before it is undertaken the surgeon should have mastered its technique.

By technique one does not necessarily mean the various steps in the operative procedure, since these are easily learned and have been very adequately illustrated. Technique implies much more than that: it connotes the manner in which the surgeon handles the tissues. It took many years for the surgeon to realize the significance of Lister's antiseptic system, which as it developed emphasized two points. These were: first, to prevent the access of infectious organisms to a wound, and, second, to do as little harm as possible to the tissues, since they could cope with only minimum contamination. It has taken less time for us to realize what Halsted taught, namely, the need for gentle, meticulous handling of tissues, careful hemostasis, and the use of fine, non-absorbable sutures. The general surgeon has, perhaps, been somewhat slow to adopt the Halsted technique. However, two surgical specialties developed to their present degree of perfection to a great extent because Halsted's teachings enabled the surgeon to carry out procedures previously thought impossible to accomplish. These specialties are neurosurgery and plastic surgery. There is no doubt but that vascular surgery, likewise, owes such of its success to the careful techniques inaugurated by Halsted.

Verdan has given us some encouragement in the pursuit of this problem and his method of primary repair by relaxation of the sutured tendon by transfixion through an area away from the site of repair, careful apposition of the tendon ends with a few fine sutures, and unroofing of the fibrous canal over the area point up the essentials of tendon repair; adequate relaxation of the site of repair, minimum disturbance of the tendon by suture and handling, and provision for the site of repair to lie in soft areolar and fatty tissue from which the initial elements of healing and blood supply must come. This is the same goal that Bunnell attempted to reach with his pull-out wires. More recently, an Austrian surgeon, Bsteh[a], has developed a technique along the same principles. This surgeon brings the divided tendon ends together by traction on the proximal stumps in the palm well away from the site of division and then transfixes them with a stainless-steel hypodermic needle which is pushed into the periosteum of the metacarpal. Only one or two fine sutures are placed in the peritenoneum of the site of division. The fibrous tendon sheath at the site of injury is relaxed by lateral incisions.

A method described some years ago has proved successful in a number of instances and it, too, follows these principles: relaxation by sutures placed some distance from the site of division, accurate apposition of tendon ends with a few fine sutures, and excision of fibrous sheath to leave a window over the area of healing. This method is not perfect because it entails perhaps more handling of the tendon than necessary. When it fails, the failure is probably due to tendon irritation at the site of repair.

The plea I should like to make is that we do not take a completely pessimistic or defeatist attitude toward primary repair in this critical zone. It is a most frequent site for tendon division and where suitable cases present themselves, it would be most advantageous to the patient if primary repair could be carried out with reasonable assurance of success. However, only by constantly striving to better our technique, by following the example of the plastic surgeon, and by observing the principles of tendon repair can we obtain this end.

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Dr. J. William Littler, New York, N. Y.: It is obvious that Dr. Verdan has developed a technique of great value for this difficult problem. Despite a limited experience with primary tendon repair my frequent encounters with the secondary problem make me appreciate his contribution all the more. With his technique more early tendon repairs can be performed with more optimism than heretofore.

A better understanding of the retinacular system of palm and digits is needed in tendon surgery. A full-length fibrous sheath is not required for good tendon function. Two strategically placed pulleys fulfill the mechanical demand and prevent tendon prolapse at the metacarpophalangeal and interphalangeal levels. These need be no wider than one-fourth of an inch and should, if possible, be left at the base of the proximal phalanx and at the center of the middle phalanx. The remainder of the fibrous sheath may be discarded. This is especially important at the site of injury and repair. However, in Dr. Verdan's repair enough of the fibrous sheath must be left for proximal transfixion of the tendon.

Let us hope, that with more attention to Dr. Verdan's refined method more of us can approach his results.

Dr. Joseph H. Boyes, Los Angeles, California: This paper points out something that Dr. Michael Mason brought out some years ago: the importance of excision of the sheath at the point of primary repair. It is commonly believed that a flexor tendon must have a tendon sheath in order to function, but this is not necessarily true. There are many tendons in the body that do not have sheaths. Excision of the sheath allows a blood supply to be provided. The same thing is true of tendon grafts. If a tendon graft in the finger is to survive, it must have a blood supply. If a tendon is put through the sheath it will die. Gonzalez showed that by putting polyethylene around the tendon the blood supply was cut off. I believe that the blood supply for a repaired tendon comes from the contiguous soft tissues and I make it a point to excise all the sheath except at the two sites mentioned by Dr. Littler. I am not convinced that relaxation of the tendon is obtained by transfixion. In about twenty primary repairs done with simple suture and with no transfixion I believe we have had only one or two complete failures. Our percentage of secondary tenolyses was not quite so high as Dr. Verdan's.

Dr. Verdan (closing): I am very happy that I found a positive echo in the discussions of Dr. Mason, Dr. Littler, and Dr. Boyes. I should like to insist on what I said at the beginning of my paper: one who is capable of making a graft of a flexor tendon is also capable of making a primary repair. You can say the same thing in a negative way: one who is not capable of making a tendon graft will not be capable of making a primary repair. I think we have to be very prudent in doing this primary repair.

Transplantation of the Index Finger in Congenital Absence of the Thumb

BY EDUARDO ZANCOLLI, M.D., BUENOS AIRES, ARGENTINA

From the Enrique and Ricardo Foundation, Buenos Aires

Digital transplantation to replace the thumb in congenital absence of the thumb poses a different problem from that presented by loss of the thumb because of trauma. In the congenital deformity there are no thenar muscles and no carpometacarpal joint, with the result that it is more difficult to obtain opposition of the transplanted digit. It is the purpose of this paper to describe an operation that was used to transplant the index finger to substitute for the thumb in a ten-year-old child with congenital absence of this digit.

In the technique to be described I followed the general and well known principles for transplantation of a finger. The first interdigital cleft was placed at the level of the proximal interphalangeal joint of the index finger to preserve as nearly a normal appearance of the hand as possible and to avoid making the new thumb appear too long.

To provide opposition of the new thumb the following procedures were

FIG. 1

FIG. 2

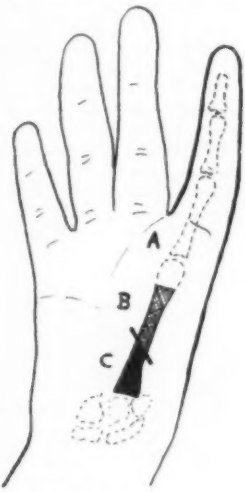

FIG. 3

Fig. 1: View of the palmar surface of the hand outlining the palmar flap, *A*, which is used to form the principal part of the first interdigital cleft. A portion of the skin of the index finger (cross-hatched area) is resected to bring the commissure into proper position.

Fig. 2: View of the dorsum of the hand showing the dorsal flap, *B*. This flap is transposed into the newly formed first interdigital cleft to lie distal to the palmar flap, as shown in Fig. 7. This flap must be planned so that it has a sufficiently wide base to provide an adequate blood supply and avoid necrosis. The bayonet-shaped incision outlined on the radial side of the wrist was used to resect a portion of the extensor digitorum communis tendon to the index finger and to shorten the tendon of the extensor indicis proprius after the index finger had been transplanted to its new position.

Fig. 3: Diagram illustrating the fashion in which the middle third of the index metacarpal was resected, *B*. The three phalanges and the head of the metacarpal of the index finger, *A*, are then transplanted to form the new thumb. Half of the proximal phalanx is left *in situ* to fill out the commissure between the new thumb and the long finger. The index finger is mobilized by ligation of the digital vessels going to the long finger and division of the transverse metacarpal ligament.

carried out: The middle third of the metacarpal of the index finger was resected and the metacarpophalangeal joint of this finger was utilized as the carpometacarpal joint of the new thumb. This was accomplished by rotating the new thumb

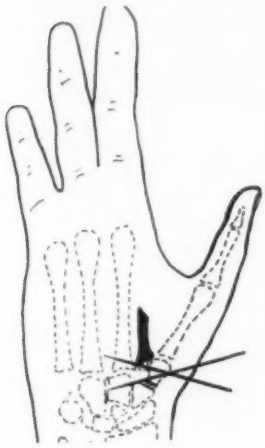

FIG. 4

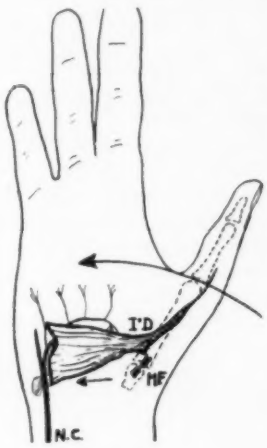

FIG. 5

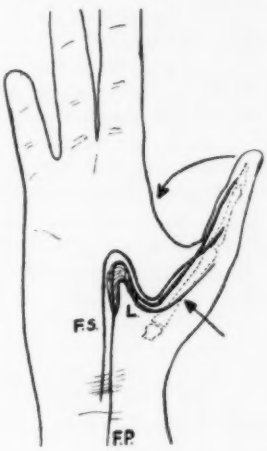

FIG. 6

Fig. 4: Diagram illustrating the method of fixing the index metacarpal to the carpal bones. A bed is prepared in an appropriate location on the carpal bones and the neck of the metacarpal is fixed to this bed by crossed Kirschner wires. The metacarpal head is so fixed that the new thumb is abducted from the palm and rotated into opposition.

Fig. 5: Transplantation of the first dorsal interosseous muscle: The origin of the first dorsal interosseous muscle is detached and moved subcutaneously to the hypothenar eminence, preserving its innervation and blood supply. The insertion of this muscle on the base of the first phalange is divided, leaving its attachment to the lateral band of the extensor mechanism of the index finger intact.

Fig. 6: Diagram illustrating how the courses of the tendons of the flexor digitorum profundus, *F.P.*, and of the flexor digitorum sublimis, *F.S.*, muscles of the new thumb, as well as the course of the first lumbrical muscle, *L*, are changed by transplantation. The angulation of the flexor tendons did not impair function since good digital flexion was soon regained. Initially, a "claw" deformity of the new thumb developed owing to relaxation of its intrinsic muscles, but this deformity disappeared as these muscles adapted to their new position.

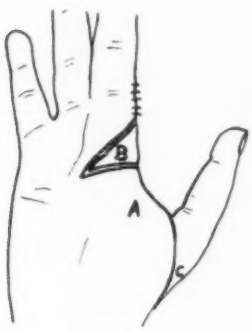

FIG. 7

FIG. 8-A FIG. 8-B FIG. 8-C

Fig. 7: Diagram illustrating the position of the palmar and dorsal flaps. In this case partial necrosis of the dorsal flap occurred owing to excessive dissection and a pedicle that was too narrow.

Figs. 8-A through 8-C: The new "thumb".

Fig. 8-A: In maximum extension.

Fig. 8-B: Opposed to the long finger.

Fig. 8-C: Opposed to the little finger. It is apparent that the transplanted first dorsal interosseous muscle is functioning well as an opponens muscle.

into the position of opposition and abduction, maintaining it in this position by fusing the head of the index metacarpal to the carpal bones. In addition, the collateral ligaments on the radial side of the index metacarpal joint were resected to permit more rotation to take place in this joint. The transverse fibers of the extensor mechanism at the level of the index metacarpophalangeal joint were maintained intact.

To provide motor power for opposition the origin of the first dorsal interosseous muscle was transplanted to the hytpohenar eminence, keeping the nerve and blood supply to the muscle intact (Fig. 5).

It is now two years since the procedure was performed. The patient at the present time uses his new thumb well and has a normal sensation. He is now able to do such things as hold a pen in correct opposition for writing with the right hand and to button, which he was unable to do prior to the procedure.

The procedure described here is presented as a practical method for improving function of the hand in congenital absence of the thumb (Figs. 1 through 8-C).

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The Hand in Leprosy

A Seven-Year Clinical Study

Part I. General Aspects of Leprosy

BY DANIEL C. RIORDAN, M.D., NEW ORLEANS, LOUISIANA

This presentation will deal with the unique problems of reconstruction of deformities of the hand in leprosy. The study was done over a seven-year period, beginning in 1949 when I was appointed Orthopaedic Consultant (Hand Surgery) to the United States Public Health Service Hospital at Carville, Louisiana. By way of background, it should be explained that I had my general surgical and orthopaedic training prior to World War II at the hospital in which Sterling Bunnell did all of his extensive hand surgery. During World War II I spent nearly two and one-half years at Valley Forge General Hospital doing both orthopaedic and hand surgery the entire time. During that period of time I had rather extensive experience with hand deformities resulting from trauma and nerve lesions, and it was with this experience that I met the problems of hand deformities in leprosy.

It soon became apparent, however, that the average experience in dealing with problems of hand surgery would not give the answer in a great many cases of hand deformities as a result of leprosy. The main reason for this was probably owing to the fact that the majority of patients first seen and treated had deformities that had been present for five, ten, fifteen, and even twenty years before any treatment had been given. Another problem was that no one had ever done any hand surgery on patients living in the National Leprosarium, and there was considerable doubt in the patients' minds whether they wished to consent to any surgery at all. Thus, it was only those patients with the most severe hand deformities who would consent to surgery, which made the problem even more difficult. In order to understand the problems of leprosy, however, it is necessary to have some information and general background of the disease. The first part of this presentation, therefore, will consist of a brief résumé of the general aspects of leprosy.

Leprosy is a chronic, infectious disease caused by the organism *Mycobacterium leprae*, an acid-fast bacillus, which was described for the first time by Hansen in 1874. The disease, regarded as only slightly contagious in most circumstances, involves chiefly the skin and peripheral nerves. Although leprosy is world-wide in its distribution, it is an uncommon disease in the United States.

HISTORICAL BACKGROUND

Leprosy is a disease associated with antiquity. The upper regions of the Nile have often been called the cradle of leprosy; there are Egyptian records dating from the sixteenth century B.C. referring to leprosy among Negro slaves from Sudan, Africa. Leprosy is now a world-wide disease, and some authorities believe that leprosy spread throughout the world by way of the early trade routes. Leprosy manifested itself for many centuries in Asia Minor, Europe, and China. It was prevalent in the provinces contiguous to Tibet and Burma for hundreds of years, lending some support to the suggestion that it was introduced by way of the ancient trade routes. In fairly recent times, leprosy has been known to spread from China to Malaya, to the Pacific Islands, and to Australia. It was most certainly introduced to Japan from China.

There has been much confusion and controversy regarding the true nature of Biblical leprosy, and it is now generally agreed that the Hebrew word *zaraath*, in

the Old Testament, included a number of other skin diseases, such as psoriasis, as well as leprosy.

Hippocrates, writing about 400 B.C., did not describe leprosy, although Aristotle, writing about 345 B.C., did mention it. It, therefore, was present in Asia Minor and on the Greek coast at that time as only a rare disease and did not become common until about 200 B.C. Records relating to the spread of leprosy to Europe through Greece about 350 B.C. show that it was probably brought by the armies of Darius, and in Italy was brought by the army of Pompey in 62 B.C. Galen, in 180 A.D., described the incidence of leprosy in what is now Germany. By the seventh century leprosy had become so common and widespread in Western Europe that, according to Virchow, there were already 636 leprosaria in Italy and in the districts of Verdun and Maastricht. Some people believe that leprosy was introduced into Europe by the return of the Crusaders, but leprosy was prevalent in practically all parts of Europe, including England and Scotland, long before the beginning of the Crusades in the eleventh century.

TABLE I

HISTORY OF THE CLASSIFICATION OF THE TWO TYPES OF LEPROSY

Origin	Time	Name		Name
Greco-Roman	345 B.C.	Elephantiasis Satyriasis Leontiasis		Leuce
Early European	180 A.D.	Elephantiasis graecorum		Elephantiasis graecorum
Arabian	19th century	Juzam		Baras
Danielssen and Boeck	1848	Tubercular leprosy	("mixed" cases)	Neural leprosy
Mexican	1880	Lepra leonina		Lepra antonina
Hansen and Looft	1895	Nodular leprosy		Maculoanesthetic leprosy
Manila Conference	1931	Cutaneous leprosy	(including "mixed" cases)	Neural leprosy
Cairo Congress	1938	Lepromatous leprosy		Neural leprosy
Havana Congress	1948	Lepromatous leprosy	(indeterminate group)	Tuberculoid leprosy

England is supposed to have established its first leprosarium in Nottingham in 625. Ireland followed suit in 869 and Wales, in 950. By the twelfth century Scotland had its leprosarium in North Tweed, and England had added another in St. Giles, near London. Holland, Denmark, Sweden, Norway, and more accessible parts of Russia on the Baltic and on the Black Sea were invaded by leprosy as early as 1256. A house for leprosy, which is still standing, was founded in Bergen, Norway, in 1266. Between the fifteenth and sixteenth century leprosy had spread —some say by way of Scotland—to the Shetlands, Iceland, and Greenland.

The steady decline of leprosy in Europe started about the fourteenth century and the reason for this decline has been a subject of controversy over the years. It is a definite fact, however, that a report by a Royal Commission in 1547 revealed that in England most of the houses for leprosy were empty. Cases still persisted in Scotland and Ireland until 1775. The disease also became less common in other countries of Europe, but is still present in small numbers in Sweden, Norway, Germany, Spain, and Portugal. It still prevails in the Baltic provinces and southern parts of Russia and Turkey. The decline of leprosy during this time was most probably due to an increased and improved diet, as well as to some prophylactic measures, chiefly isolation. Isolation, however, cannot be credited with being the sole factor leading to the decrease of leprosy in Europe at that time.

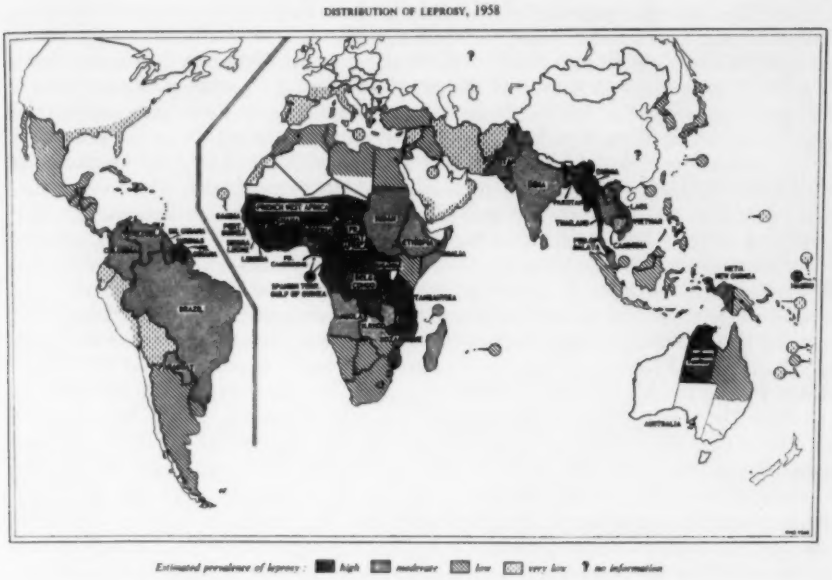

FIG. 1
Map showing the distribution of leprosy throughout the world.

At the time leprosy was decreasing in Europe it was being carried to the Western Hemisphere with the discoveries of the West Indies and the Americas. Many authorities believe that the Indians of the Western Hemisphere were free from the disease until it was carried by Columbus' soldiers and, later, the slaves from West Africa.

It is believed that leprosy existed in Mexico as early as the sixteenth century and that some of the cases appearing in the southern part of the United States came from what was originally Mexico. The earliest reference to the disease in what is now the United States was found in Romans' *Concise Natural History of East and West Florida*, published in 1776. The author reported the occurrence of leprosy in the provinces as early as 1758, and the establishment of a leprosarium near the mouth of the Mississippi River by the Spanish governor in 1766. Thus, it may be concluded that leprosy has existed in the United States for about 200 years. The disease is constantly being reintroduced, however, by immigrants. The imported cases appearing in the Atlantic Coast states have entered from Europe, Africa, and the West Indies; in the Gulf states, from Africa, the West Indies, and Mexico; in the West Coast states, from the islands of the Pacific, Asia, and Mexico; and in the North Central states of Wisconsin, Iowa, and Minnesota, from Norway. A map showing the incidence of leprosy has been maintained at the National Leprosarium in Carville, Louisiana, since 1921, when the hospital was established. It is quite apparent from this map that the highest concentration of cases is in the area of ports of entry in coastal states along the border between Mexico and the United States and in the North Central area, which has had a large influx of immigrants from Norway. Each foreign-born leprous patient cannot be said to have contracted the disease in his native country; he may have been infected by a companion after his arrival in the United States. Florida, Louisiana, and Texas are now generally described as endemic states, with California and New York having a high incidence of cases, the majority being in persons of foreign birth. Endemic cases, however, have been reported from both California and New York. In all, forty-one states and the District of Columbia have had patients diagnosed and admitted to the National Leprosarium.

In 1785, nineteen years after the opening of the first leprosarium in Louisiana, Governor Miro established, and, in 1807 abandoned, another refuge for leprous patients in that state—this time in New Orleans. In 1880 the Louisiana State Board of Health made a survey of the State and found that the incidence of leprosy was increasing. In 1894 the Indian Camp Plantation at Carville was obtained as a site for a hospital and in August of that year the State passed an act providing for the care of leprous patients. One year later the Sisters of Charity volunteered to care for the patients and have done so up to the present time. On February 1, 1921, the National Leprosarium was officially opened after the Federal Government had obtained the institution from Louisiana in 1920.

Although the United States Public Health Service Hospital at Carville had an orthopaedic consultant for about twenty years, no definitive orthopaedic surgery or reconstruction or rehabilitation program was carried out during that time. In 1947, William Reggio, who was then Medical Director and Chief of Physical Medicine and Rehabilitation Section in the Division of Hospitals of the United States Public Health Service, made a survey of the patients and the problems at Carville. He realized the patients' need for rehabilitation, and it was through his efforts that the program which was later set up was started. In 1948, my associate, Rufus H. Alldredge, was appointed Orthopaedic Consultant at Carville. A physical therapist was also appointed at that time and started treating the patients under the direction of Dr. Alldredge and, at a later date, under a physiatrist. Dr. Alldredge was instrumental in getting my appointment at Carville to help in the problems of reconstruction of the hands in the severely disabled patients, and this work was started in 1949.

ETIOLOGY

Mycobacterium leprae, the organism causing leprosy, is an acid-fast bacillus that can be isolated from nearly all cases of the disease. However, positive proof of the relationship of the organism to the disease has never been proved according to Koch's postulates: the organism has never been successfully grown on artificial

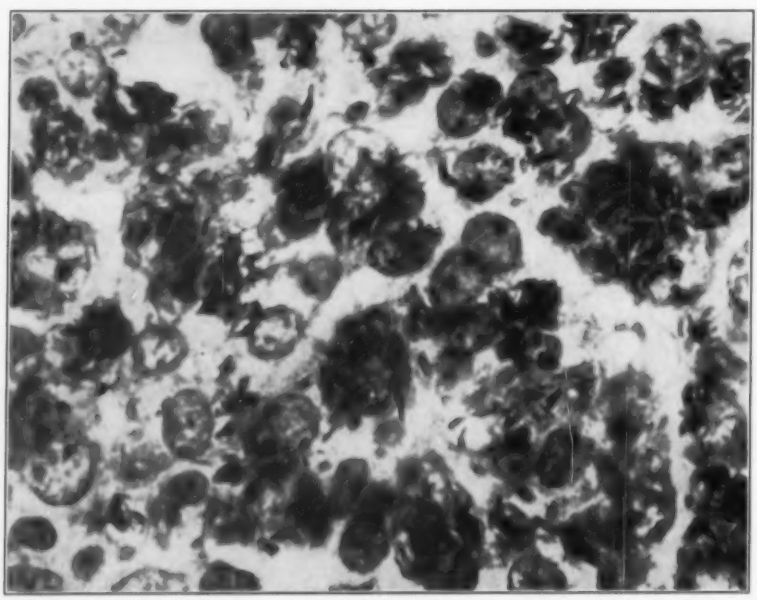

FIG. 2

Mycobacterium leprae as it appears in an acid-fast stain.

media or successfully inoculated into animals or human volunteers. On the other hand, the organism is conspicuously abundant in every case of lepromatous leprosy and, although appearing in smaller quantities, is also found in many cases of tuberculoid leprosy. Unquestionably, this would preclude any doubt about the organism's being the actual cause of leprosy.

Smears from skin scrapings or in sections from skin biopsies show that *Mycobacterium leprae* is a straight or slightly curved acid-fast rod about three to five micra long and 0.2 to 0.4 micron thick. If many bacilli occur together, they tend to be aggregated into packets not unlike bundles of cigars and in much larger aggregates with spheroid contours known as globi. The globi are thought to represent masses of bacilli that have been ingested by phagocytic reticuloendothelial cells in the course of the generalized foreign-body reaction-like process that characterizes lepromatous leprosy. It is possible that many of the bacilli may have multiplied within such cells, thus helping to account for the large numbers of bacilli in such globi.

TABLE II

THE CATEGORIES OF LEPROSY AS DEFINED AT THE FIFTH INTERNATIONAL LEPROSY CONGRESS, HAVANA, 1948

Lepromatous (malignant, or gravis) type: symbol, L.	Minimal resistance to the existence, multiplication, and dissemination of the bacilli; constant presence of large numbers of bacilli in the lesions, with a distinctive tendency to form globi; characteristic clinical manifestations in the skin, mucous membranes (especially those of the upper respiratory tract and eye), or the peripheral nerves, or both, together with involvement of other organs; regular failure to react to lepromin; pathognomonic granulomatous structure of the lesions; marked stability of type; and a tendency to progression. These cases are "infectious", or "open".
Tuberculoid (benign, or mitis) type: symbol, T.	High resistance to the existence, multiplication, and dissemination of the bacilli; bacteriologically negative as a rule or, if positive, with few bacilli except in reactional states; characteristic clinical manifestations, mainly in the skin and nerves, tending to be limited in extent and varying in degree with the reactivity of the tissue; reactivity to lepromin in a very high percentage of cases; nearly always a tuberculoid granulomatous structure in active lesions; marked stability; and a strong tendency to spontaneous regression in the absence of repeated reactions. These cases are usually "non-infectious", or "closed".
Indeterminate (undifferentiated) group: symbol, I.	Variable with respect to resistance; clinical manifestations chiefly in the skin and nerves; the skin lesions usually flat maculae, either hypochromic, erythematohypochromic, or erythematous; bacteriologically negative as a rule, or, if positive, with few bacilli; lepromin reaction usually negative or moderately positive; the lesions histologically of simple inflammatory nature; stability much less than in either of the (polar) types; and a variable tendency with regard to persistence, progression, or transformation into one of the polar types. These cases are usually "non-infectious".

There have been many attempts to transmit leprosy to human volunteers by inoculating them with fresh, presumably infective material and all have uniformly failed, except one. This exception is a debatable one—some authorities stated that it was a true case of inoculation and others stated that it was not. Mouritz and Wade described an inoculation of the infective tissue made by Arning into the skin of a Hawaiian named Keanu; a leproma developed at the site of inoculation, which suggests that the experiment was successful. The importance of the result has been questioned, however, because Keanu was known to have had leprous relatives and thus might have been infected by the usual routes. Lowe reported the simultaneous development of tuberculoid leprosy in two American Marines from Michigan. These Marines, on the same day in 1943, in the same shop in Australia during World War II, were tattooed, and leprosy developed in the tattoos. Michigan is a wholly non-endemic area, and this would seem to constitute two more examples of successful infection of human beings by inoculation.

EPIDEMIOLOGY

Diseases like leprosy and tuberculosis can be considered to be epidemic diseases and thus behave like other epidemic diseases, rising to a peak and gradually declining. In leprosy, however, the curve of the epidemic cannot be considered in terms of weeks or months, but in centuries. In this manner, the epidemic of leprosy in the British Isles lasted from 600 to 1700—a period of eleven hundred years. Contradictions and paradoxes make the epidemiology of leprosy indeed puzzling; some historians believed leprosy to be highly contagious and others believed it to be hereditary. The uncertain and probably highly variable period of incubation makes epidemiologic studies difficult to interpret. In infants the period may vary from as little as one to two years; in adults the range may be from five to ten years, or even longer. There are many factors that influence the epidemiology of leprosy, and a few of these will be considered: age, sex, race, geographical location, diet, insect vectors, climate, and inherited susceptibility.

Age

Many writers have emphasized the significance of leprosy in children. Some authorities are convinced that the great majority of persons with leprosy were infected and showed manifestations of the disease before they were fifteen years old. After the age of twenty-four there is a sharp decline in the incidence of attack. Although it is quite certain that children are very susceptible, it should be pointed out that adults are also susceptible. There are many cases of leprosy in persons whose exposure to the disease definitely occurred when they were adults. On the other hand, the occurrence of leprosy in adult attendants in leprosaria is quite rare.

Sex

There is considerable disparity in the opinions of leprosy workers the world over regarding the incidence of leprosy in relationship to the sex of the patient. The most prevalent concept is that it is twice as common among men as among women. In studies done at the National Leprosarium Hopkins and Denney, and Faget pointed out that the incidence of male and female patients was equal. In the Philippines, both Rodriguez and Lara observed that among children the incidence of disease was the same in boys as in girls, whereas in India Cochrane found that in those affected with leprosy the ratio of male to female was fifty-one to thirty-two (per 1,000) among adults and only seventy-seven to sixty-eight (per 1,000) among children.

Race

Racial susceptibility to leprosy is almost impossible to evaluate because other factors—propinquity, diet, and inherited susceptibility—cannot be separated. Yet, there are sufficient variations in incidence among different races to warrant mentioning some of them briefly. Cochrane, in India, reported that lepromatous eye lesions were three times as common among Anglo-Indians as among Indians. He also reported that laryngeal lesions occurred thirty times as often in Anglo-Indians as in Indians. In Hawaii Chung-Hoon reported the incidence of leprosy to be 70.2 per cent per 100,000 per year for the Hawaiian population and only 1.1 per cent per 100,000 per year for the Caucasian population. Part-Hawaiians showed a rate of 13.9 per cent; Filipinos, 13.6 per cent; Chinese, 4.3 per cent; and Japanese, 2.6 per cent.

Geographical Location

The distribution of leprosy throughout the world has varied dramatically. Early records show that the disease spread from India, Africa, and China to Europe, where it remained prevalent for about 500 years, or until the fifteenth cen-

FIG. 3
Male patient with lepromatous leprosy.

tury, when it gradually declined. Leprosy has remained definitely endemic only in Africa, India, Burma, South China, Japan, Central and South America, Mexico, the West Indies, the Malayan Archipelago, and many of the Pacific Islands. The incidence in these areas is sporadic, and the number of cases often varies widely in villages only a few miles apart. Recently, the introduction of new cases of leprosy into areas previously free of the disease has not, as a general rule, produced new endemic foci. For example, the immigration of Norwegians into the North Central United States in the mid-nineteenth century resulted in only a few new secondary cases. In marked contrast, however, is the island of Nauru in the South Pacific, where the arrival of one person with an open case of leprosy was followed by an epidemic in the native population.

Diet

Diet has not been shown to be a cause or to be directly responsible for the incidence of leprosy, except in a general way. It has been generally observed that leprosy is rare in areas where the diet is good, but is common in areas where the diet is inadequate. This is usually thought to mean that an inadequate diet simply increases susceptibility to infection and perhaps decreases resistance to it once infection has been established. Jonathan Hutchinson believed that imperfectly salted and partially spoiled fish was the cause of leprous infection; he devoted an entire book to the subject in 1906. This view, however, was not widely accepted and is now completely discredited. Although it is true that leprosy is endemic in those countries where fish forms a large part of the national diet, fish-eating in itself is not contributory to infection with the disease. It has been pointed out that leprosy is more prevalent in those parts of India and China where rice is the staple grain than in areas where other cereals and different diets prevail.

Insect Vectors

Many types of insects have been named at various times as being possible vectors of leprosy. Nevertheless, there is neither proof nor documentary evidence to support the concept that bedbugs, fleas, house flies, mosquitoes, cockroaches, or other insects are to blame for the spread of the disease.

Climate

So many other factors are part of the general picture of leprosy that it has been impossible to separate and evaluate the factor of climate. It does appear, however, that leprosy is not readily transmissible in temperate zones—where, conversely, the weather is intemperate—except in Japan, which has a high incidence of the disease. Generally speaking, however, it can be said that leprosy is a

disease peculiar, at the present time, to tropical and subtropical climates.

Inheritance of Susceptibility

The concept that leprosy was transmitted solely by inheritance was vigorously introduced by Danielssen and Boeck in 1848, and politely discounted in 1895 by Hansen, who demonstrated the causative organism of the disease. The theory of inherited susceptibility, however, has remained popular even into this century, based, no doubt, on the fact that leprosy does have a tendency to occur more frequently among blood relatives than among those persons related only by marriage. Aycock defended this view of inherited susceptibility to infection and defended it until his death in 1951, at which time he was involved in a genealogical study of infected families in Hawaii. Cochrane, however, is skeptical of the theory of familial susceptibility; he observed that the percentage of infection among children exposed to the disease is approximately the same, whether a blood relative or some other occupant of the same household was the source of infection. At the moment it appears that the many discrepancies in the epidemiology of leprosy could be explained by Aycock's view of inherited susceptibility, but further elaboration on this aspect of epidemiology must be done before it is acceptable.

CLASSIFICATION

From its first reference in the Bible, and for many centuries thereafter, the nomenclature of leprosy was so varied that history presents a most confusing picture; many of the diseases classified as leprosy in those times were actually some other skin disease. The word leprosy, from the Greek word *lepra*, originally meant any scaly disease or some form of psoriasis. The association of the words leprosy and *lepra* with misconceptions of contagion has stemmed, most probably, from the Bible and has engendered a fear of the disease that has been carried down to the present time. Danielssen and Boeck, in 1848, named the two individual forms of the disease "nodular" and "anesthetic" and further described a combination of these two types. Their criterion for the classification of individual cases of the disease was the anatomical location of the principal lesions; the nodular form was found in the skin, the anesthetic form in the nerves, and the mixed form was present in both skin and nerves. This simple anatomical classification, although it was misleading, influenced all concepts of the disease for over fifty years. The classifications persisted despite the refutation written by Hansen and Looft in 1895 and published in Norwegian, German, and English. Hansen and Looft called the two types of the disease *lepra tuberosa* and *lepra maculoanesthetica* or, in English, nodular leprosy and maculoanesthetic leprosy, and deleted the term mixed leprosy since each of the two forms of the disease almost always involves both skin and nerves. In 1931, at the Leonard Wood Memorial Conference on Leprosy in Manila, by general agreement, an official designation was given to the two forms of leprosy: cutaneous and neural, respectively, with a mixed form of leprosy as a subvariety of the cutaneous form. These designations agreed with those of Hansen and Looft, but because they were so uncomplicated and descriptive, they were paradoxically misleading: there was a tendency to disregard the fact that the two types were mutually incompatible and were not to be classified exclusively on an anatomical basis. In 1938, at the International Congress on Leprosy in Cairo, the term cutaneous was abandoned in favor of lepromatous, but the term neural was retained. In 1948, at the Fifth International Congress on Leprosy in Havana, the term lepromatous leprosy was retained, but neural was changed to tuberculoid leprosy. At the same time it was agreed that there was still another group of cases distinct from either type just mentioned that should be called indeterminate, since they could not be classified as either lepromatous or tuberculoid leprosy.

Because literature, both Biblical and historical, has given leprosy the stigma of contagion, even the word itself evokes an unreasonable fear. Therefore, at the

TABLE III

DIAGNOSTIC ESSENTIALS FOR THE CLASSIFICATION OF LEPROSY

Criterion	Lepromatous Type	Tuberculoid Type	Indeterminate
Bacilli usually found at skin-lesion bacterioscopy	Abundant	Absent, except in reactions	Present or absent
Nerve thickening	Slight, slow, and symmetrical	Early, marked, and asymmetrical	Variable
Anesthesia	Independent of skin lesions	Coextensive with skin lesions	Variable
Histology of lesions	Macrophages and histiocytes	Epithelioid-cell tubercles and lymphocytes	Banal, round-cell infiltrate
Lepromin reaction	Negative	Positive	Negative or weak-positive

Havana Congress in 1948 a committee proposed that the name be changed to Hansen's disease. The recommendation, however, was not passed, and leprosy was retained as the official designation of the disease, although it was suggested that the word leper be abandoned when describing leprous patients.

DIAGNOSIS

The diagnosis of leprosy in early cases is frequently quite difficult, even for the alert physician, especially in the absence of any readily apparent nerve involvement. Advanced cases of leprosy, however, are usually not so difficult to prove, owing to the combination of skin lesions and nerve lesions which, in itself, should arouse suspicion of the disease. It is frequently the dermatologist who gets the chance to make an early diagnosis since the majority of the early symptoms are those involving the skin. The lesions may appear as pale spots or blotches, but they are occasionally hyperpigmented or reddened and closely resemble the lesions of pityriasis or *Tinea circinata*. The central clearing and scaliness may be present, although scaliness is uncommon in leprosy, and itching almost never occurs. Vitiligo may seem to be present but it is not true vitiligo since actual depigmentation never occurs in leprosy, except in scars, although the lesions are sometimes hypopigmented. Seborrheic dermatitis may be simulated, but it is rarely limited to just those sites usually involved by seborrhea. Pityriasis rosea, although spurious, is imitated so minutely that even an expert diagnostician may be confused; one must be alert to the signs of differentiation—associated anesthesia, thickening of the nerves, and, most usually, absence of itching. At times, the pale facial maculae of impetigo sicca are difficult to distinguish from the maculae of leprosy; even the methacholine sweating test is not helpful since anesthesia is slow to develop on the face and sweating is usually present. (The histamine test, however, may be helpful in this situation.) When the maculae of impetigo are present in Hawaiians, one must be aware that there is a racial tendency in these people to lesions almost identical with those of leprosy. The smooth hypopigmented form of psoriasis, common in Oriental patients, also closely simulates leprosy, but the scaly silvery type of psoriasis is clearly not a manifestation of leprosy. The early lesions of leprosy may also simulate lupus erythematosus, but this resemblance is dispelled by fully developed lesions of discoid lupus erythematosus, which are not found in leprosy.

Lumps and nodules are frequently present and often precipitate a misdiagnosis of urticaria or erythema nodosum. Early lesions of lepromatous leprosy often resemble urticarial wheals, and erythema nodosum may occur as part of the febrile allergic flare-up—the lepra reaction. A common point of differentiation in these two lesions is the absence of itching in leprosy. Another characteristic is that on finger pressure the lepromata do not buttonhole or drop easily into the underlying fatty tissue. Biopsy, however, should give the proper diagnosis in this type of case.

Sarcoidosis should be mentioned here because the histological changes seen both in Boeck's sarcoid and in tuberculoid leprosy are so alike that a clinical misdiagnosis may be erroneously confirmed histologically. Rather than biopsy, testing the skin lesions for signs of denervation is probably the most reliable way to distinguish tuberculoid leprosy from sarcoidosis. In leprosy denervation of the skin lesions will usually be present in all but some early facial lesions. This is not the case in non-leprous sarcoid, where there is never any denervation. Another common misdiagnosis is that of erysipelas. Although this disease may exactly counterfeit early tuberculoid leprosy, a pertinent differentiation is that patients with tuberculoid leprosy do not have the high fever that usually goes with erysipelas.

Other signs may be found, such as thinning or loss of eyebrows and, occasionally, loss of eyelashes, but these are usually late changes. Although many eye lesions appear in lepromatous leprosy they are usually late findings and consist of episcleritis, pannus formation, keratitis, and iridocyclitis. One should be vigilant in tuberculoid leprosy and, occasionally, in leprous children for possible widening of the palpebral fissure by slight ptosis of the lower lid. Leprous involvement of the larynx will cause hoarseness, but this is a late manifestation of the disease.

Many reported neurological changes appear early in the sequence of leprosy; numbness in the skin in the course of a peripheral nerve or in a circular or glove fashion may actually be present. I have never found any article in the literature that described a true anatomical lesion in which there is an extensive circular glove or stocking loss of sensation in any disease other than leprosy. Textbooks of neurology will state that any person with a peripheral-nerve involvement having a circular loss of sensation is usually hysterical except for the patient with advanced diabetes, which is accompanied by very limited stocking or glove loss of sensation. In leprosy, however, one may see a mild ulnar-nerve paralysis with loss of sensation involving the entire circumference of a hand and forearm clear up to the elbow or, at times, even to the axilla. The same is true of the foot, and it is not uncommon to find peroneal paralysis associated with a complete loss of sensation up to the mid-thigh. Localized loss of sensation, such as in the distribution of the lateral femoral cutaneous nerve, may be confused with so-called meralgia paresthetica. Any case of peripheral neuritis accompanied by nodular thickening of the nerve trunk is usually equated with leprosy. It is extremely rare for any other neurological disease to cause thickening and enlargement of major nerve trunks. It should also be remembered that leprosy rarely causes loss of tendon reflexes, except in the most advanced degrees of nerve paralysis. The appearance of ulcers on the plantar aspect of the feet may sometimes be confused with tabes dorsalis or syringomyelia. In the case of leprosy, the ulcer is not a leprous lesion but is simply a trophic lesion resulting from loss of sensation. Without the protective device of burning pain on compression, there is loss of circulation on a mechanical basis, owing to pressure; ultimately, this leads to tissue necrosis.

Syringomyelia may be confused with leprosy because of the loss of pain and temperature sensation, but with the preservation of touch. It has been said that the earliest nerve involvement in leprosy is the loss of pain and temperature perception. Later, the loss of touch accompanies this involvement; in leprosy the signs will usually be definitely peripheral in nature, whereas in syringomyelia they will be central and will extend up to the center of the mid-line of the body. The histamine test or the sweating test, which should help to differentiate these diseases, will be described later. The diagnosis of Bell's palsy or facial paralysis can usually be ruled out since leprosy is usually accompanied by other findings.

Traumatic neuritis resulting from an injury to the ulnar nerve can usually be distinguished from leprosy by the lack of other manifestations of the latter entity, especially thickening of the nerve.

Another neurological condition which I have not yet found described as closely simulating leprosy is Charcot-Marie-Tooth disease. This neurological disorder is characterized by a progressive peroneal atrophy and, later, paralysis which

may be so extensive as to involve both the peroneal and tibial nerves in the legs up to the knee and, occasionally, the lower third of the thigh. It never goes higher than this, however, and even with extensive paralysis of the limbs up to the lower part of the thigh there is rarely any loss of sensation, except in spotty areas. In leprosy, however, involvement of this extent would usually be associated with equally or more extensive loss of sensation, accompanying changes of the skin, and complete loss of all sensory modalities. In the upper extremity Charcot-Marie-Tooth disease produces an intrinsic paralysis of median and ulnar nerves in patients with advanced disease and usually does not involve the radial nerve. Even in an advanced case of Charcot-Marie-Tooth disease, where there is complete intrinsic median and ulnar-nerve paralysis, the sensory loss is again limited and spotty, whereas in leprosy it is frequently extensive, glovelike in nature, and may advance up to the elbow or even to the axilla. I have personally traced several patients—eventually diagnosed as having leprosy—who, when they were ten or twelve years old, were diagnosed as having Charcot-Marie-Tooth disease and were consequently operated on. These patients had the typical findings of advanced Charcot-Marie-Tooth disease, and the surgery consisted in triple arthrodeses and tendon transfers. Other signs typical of leprosy developed in these patients ten to fifteen or more years later, when they were seen and treated in the National Leprosarium after the proper diagnosis was established. It therefore seems apparent that orthopaedists who see cases of Charcot-Marie-Tooth disease should be aware that this disease more closely simulates leprosy than any other neurological disease and that the main differentiating factor is the extensive loss of sensation found in leprosy but absent or only spotty in Charcot-Marie-Tooth disease.

Tables III and IV show the essentials of classification and the difference between the two major types of leprosy. By way of summary, the diagnosis of leprosy should be made when acid-fast bacilli morphologically consistent with *Mycobacterium leprae* are found in sites other than open ulcers—smegma, nasal scrapings, or skin surfaces, in which case they may be assumed to be that organism. A confirmatory or equivalent criterion is evidence of nerve damage that cannot reasonably be attributed to trauma or to non-leprous peripheral neuritis. In lepromatous leprosy nerve damage is not always present in early cases, but bacilli will almost always be found in the skin lesions. In tuberculoid leprosy, however, nerve damage is usually explicit rather early, whereas bacilli are absent or difficult to find except during reactions. The simplest and most widely used method of searching for bacilli in skin lesions is Wade's "scraped incision" method in which the point of a scalpel or the corner of a single-edged razor blade is inserted into the skin over the border of a lesion to a depth of from two to three millimeters. The skin is first tightly compressed between thumb and finger to numb the area partially and to minimize the bleeding. The razor blade is then rotated on an axis perpendicular to the skin so that the edge of the blade scrapes against one side of the cut and removes a small drop of tissue fluid and cells. The material thus obtained by the scraping is placed upon a clean microscope slide, allowed to dry, and then stained by the Ziehl-Neelsen method. Another method is that which Souza-Araujo described and which he believes to be more sensitive than Wade's test. This method consists in pinching a fold of skin in a large curved Pean hysterectomy hemostat and then puncturing it deeply in four points with a large needle. The lymph that exudes from the tissues is collected and dried, then stained by the Ziehl-Neelsen method. For the deep biopsy specimens the unmodified Ziehl-Neelsen stain is not so satisfactory as the Faraco modification of the stain. Some feel that Wade's modification of the Faraco stain is even better. In tuberculoid lesions organisms are difficult to demonstrate, but careful search of serial sections will often lead to the discovery of a few bacilli.

The incidence of acid-fast bacilli in nasal scrapings from patients with leprosy is quite high but, since normally there are acid-fast bacilli in mucous membranes

of the nose, and, since they are quite similar to *Mycobacterium leprae*, these positive scrapings from the nose alone should only be regarded as one aspect of the diagnosis. The diagnostic tests for leprosy should always be accompanied by a close search for nerve damage and for asymmetry of the face and hands, a manifestation caused by weakness or atrophy of the muscles. Palpation of the neck, the region of the ulnar nerve at the elbow, or the peroneal nerve at the knee for thickening or for nodular, stiffened, or tender nerve trunks should be done. Asymmetrical thickening of a nerve is usually significant and, if the thickening is marked, should be considered characteristic of leprosy. It should be remembered that loss of touch in a skin lesion is almost always diagnostic of leprosy, whereas preservation of tactile sensation is of no significance. Loss of pain and temperature sensation should always be viewed with suspicion as far as leprosy is concerned.

The histamine test described in 1931 by Rodriguez and Plantilla is a sensitive and objective test for intracutaneous-nerve damage. Since reaction is indicated by a lack of redness about the area of the treated skin this test, useful in Caucasians, is difficult to interpret in the pigmented skin of an Indian or Oriental and almost impossible to identify in a Negroid skin. The technique is to place one drop of 1:1000 solution of histamine diphosphate on the skin inside and one drop outside the area of the lesion to be tested. A superficial pinprick is then made through each drop. A wheal will form at each puncture site; in the normal skin a red flare will form about the wheal. If intracutaneous nerves have been destroyed, there will be no red flare. This is true in leprosy and leprosy alone. In persons with syringomyelia, as in normal persons, both wheals will be surrounded by a flare. In those leprous patients with dermographia, simple scratching of the skin without the use of histamine will demonstrate the positive test; the resulting redness will stop abruptly at the border of the involved skin.

The methacholine sweating test is particularly useful in patients with deeply pigmented skin. The technique of this test is to use 0.1 cubic centimeter of 1 per cent solution of methacholine chloride injected intradermally either at the border of the lesion or both inside and outside the lesion. This is done after first painting the area with Minor's solution (2 per cent iodine and 10 per cent castor oil in absolute alcohol). A powder-blower is then used to dust the area with powdered starch. Each sweat droplet which then appears moistens the dry iodine-starch combination and turns it a deep blue. The lack of sweating, as detected by this method, in the lesions of leprosy is probably an earlier manifestation of nerve damage than is the occurrence of thermal anesthesia. As previously mentioned, in facial lesions there may be a false-positive anhidrosis, because in this area the absence of sweating may not manifest itself until late in the disease.

LEPROMATOUS LEPROSY

Lepromatous leprosy is generally considered to be the progressive or malignant form of the disease. It is characterized by nodules throughout the skin, considerable thickening of the involved skin, low resistance of the host, and widespread involvement of the stricken person. Many think of lepromatous leprosy as a process in which the human host treats the leprosy bacilli like fatty foreign bodies, storing them in cells of the reticuloendothelial system. The bacilli can be demonstrated in practically all tissues of the body, but true pathological lesions are found only in the skin, the mucous membranes of the respiratory tract, the testicles, the anterior chambers of the eyes, and the nerves.

Skin Lesions

Although no attempt is made to classify the skin lesions of leprosy specifically, it can be said, in a general way, that they fall into four main categories: macular, diffusely infiltrative, nodular, and the pure diffuse or spotted leprosy of Lucio.

The maculae of lepromatous leprosy are hypopigmented, rather ill-defined

TABLE IV

RELATIVE CHARACTERISTICS OF LEPROMATOUS LEPROSY AND TUBERCULOID LEPROSY

Characteristic	Lepromatous Form	Tuberculoid Form
Clinical		
Site of election	Skin primarily, nerves secondarily	Nerves primarily, skin secondarily
Visceral lesions	Reticuloendothelial system and testes	Rare
Mucosal lesions	Early and frequent, mainly in the larnyx	Rare, and nose only
Eye lesions	Frequent and late	Rare
Pale maculae	Infrequent	Frequent
Annular plaques	Infrequent	Frequent
Erythema multiforme	In reactions	Rare
Fever	In reactions	Rare
Eyebrow alopecia	Frequent	None
Gynecomastia	Frequent and late	None
Symmetry of lesions	Frequent	Rare
Nerve enlargement	Slow and symmetrical	Rapid and asymmetrical
Nerve damage	Slow	Rapid
Histological		
General pattern	Xanthoma-like	Sarcoid-like
Lymphocytes	Few	Numerous
Giant cells	Occasional (foreign-body or Touton)	Frequent (Langhans type)
Lipoid	Abundant	Minimum
Necrosis	Rare	Rare, except in nerves
Nerve changes	Fibrosis; structure well preserved	Normal architecture obliterated early
Visceral amyloidosis	Frequent	None
Bacterioscopic		
Acid-fast bacilli	Always abundant	Rare, except in reactions
Special Tests		
Lepromin reaction	Mitsuda negative	Mitsuda positive
Tests for syphilis	40 to 60% positive	90 to 98% negative
Hyperglobulinemia	Frequent	Rare
Sedimentation rate	Elevated, especially during reactions	Normal
Course		
Untreated	Progression	Spontaneous recovery, often
Intercurrent tuberculosis	Common	Rare
Treated	Slow regression	More rapid regression
Transition to other type	Rare, except perhaps under treatment	Unusual, but does occur in severe and relapsing (borderline) cases

at the borders, and are usually multiple and widespread. They are sometimes slightly erythematous and slightly infiltrated. Their surface is usually smooth. Sensation is usually present. Microscopically, they almost always show abundant bacilli, dense aggregations of spindle-shaped lipid-filled histiocytes, vacuolated lepra cells, and, occasionally, Touton or foreign-body giant cells.

Diffusely infiltrative lepromatous leprosy is probably just an advanced stage of the macular form. The skin is diffusely thickened and has the texture of an orange peel. Normal skin folds disappear in a way which may resemble diffuse lymphoblastoma of the skin.

Nodular lepromatous leprosy, the most common variety in the United States, is characterized by large nodules which usually are reddened and elevated and have rather sharp margins. These nodules rarely ulcerate, but, occasionally, during a lepra reaction, they may do so superficially. These nodular growths are not true inflammatory granulomata, but probably represent foreign-body granulomata in which the bacilli grow and multiply. The location of the nodules is typically in

areas where the subcutaneous tissue is abundant; they seem to avoid warmer areas, such as the axilla and groin and areas where the subcutaneous tissue is dense—scalp, palms, soles, and the intermammary fold. Ears, nose, lips, chin, elbows, buttocks, and knees are the most common sites of involvement. The growth of the nodules does not destroy the normal creases in the skin but leads to the deeply furrowed brow of the typical leonine face seen in most textbooks.

The nerve involvement may not coincide with the extent of the lesions in this type of leprosy; it is frequently not associated with the lesion itself and may even be quite widespread. Microscopically, both diffusely infiltrative and nodular lepromatous leprosy show the typical lepromatous granulation tissue.

FIG. 4
Enlargement of the ulnar nerve.

The diffuse or spotted leprosy of Lucio (lazarine leprosy), apparently a rare form of the disease, is seen mostly in central Mexico and Costa Rica where it constitutes 30 to 60 per cent of the cases of lepromatous leprosy. The patient with this form of leprosy exhibits neither maculae nor lepromata; instead, there are recurrent crops of flaccid bullae on the extremities, which undergo superficial gangrene and then heal with scarring. This type of leprosy is considered the most progressive and malignant form of the disease; the untreated patient often dies within the comparatively short period of six or seven years. A patient with an ordinary case of lepromatous leprosy, on the other hand, usually survives fifteen or more years without treatment. The cutaneous infiltration in the diffuse form of the disease involves practically all of the skin but makes it smooth and stiff, without the distortion or disfigurement characteristic of the other types of lepromatous leprosy.

Lesions of the Mucous Membrane

The mucous membranes of the upper respiratory tract are most often involved in patients with lepromatous leprosy. Acid-fast bacilli are usually abundant in the nasal smears of practically every patient with advanced lepromatous leprosy. If left untreated there may be ulceration and perforation or destruction of the cartilaginous septum of the nose, usually resulting in severe facial deformity. Ulceration of the nasopharynx, palate, or pharynx may occur, and occasionally the larynx is involved. Before sulfone, this last involvement usually led to laryngeal

obstruction and subsequent tracheotomy. With present-day treatment, however, most of these complications have been prevented, and tracheotomy is now a rarity.

Visceral Lesions

The testicle is most frequently involved in lepromatous leprosy, with resultant hyaline degeneration of the tubular epithelium. Lepromatous infiltration of the testes is actually observed in autopsy material, with destruction of the interstitial tissues and fibrosis in the advanced cases, which probably contributes to the occasional occurrence of gynecomastia. The spleen, lymph nodes, and bone marrow all contain lepra cells and bacteria, but only rarely are these lesions clinically significant.

Secondary amyloidosis of the kidneys, spleen, liver, and adrenal glands is a most frequent accompaniment of advanced lepromatous leprosy. The incidence of amyloid disease in leprosy is considerably higher than that of amyloidosis in tuberculosis or other chronic diseases. The liver may sometimes present evidence of miliary hepatic lepromata and is occasionally enlarged and palpable; in the Lucio form of leprosy it is regularly enlarged. Liver dysfunction is not a clinical feature of leprosy, yet hyperglobulinemia, elevation of gamma globulin, a positive cephalin-cholesterol flocculation test, and a positive serum colloidal test occur regularly. Gynecomastia, seen commonly in advanced cases, is probably due partly to the involvement of the testicle and partly to the deficient destruction of estrogen by the liver. It would thus seem that lepromatous leprosy does involve the function of the liver.

Eye Lesions

The anterior chamber of the eye is involved frequently in advanced cases of lepromatous leprosy, but there is no set pattern of appearance. One may see either superficial or deep keratitis, with or without corneal ulceration, or focal or diffuse episcleritis. Pannus formation may be present and may extend clear across the cornea. Acute or chronic iritis or iridocyclitis is often present, and in advanced cases of the disease, glaucoma may result from the eye involvement. Part of the damage to the cornea may be due to the lagophthalmos caused by involvement of the facial nerve and its branch supplying the eyelid muscles and by loss in sensation in the cornea. The inability to close the eyelid tightly or to blink normally and thus keep the cornea moistened may lead to ulcers and eventual scarring and damage of the cornea. Blindness was an extremely common complication in the past when sulfone was not available.

Nerve Lesions

As has been mentioned previously, the bacilli are found in the peripheral nerves even at the beginning of the disease. The irritative fibroblastic reaction that the bacilli cause in the nerve usually advances slowly except during a lepra reaction, when it may be accelerated. In a general way, the nerve involvement of lepromatous leprosy tends to be bilateral and symmetrical. The extent of the nerve involvement does not parallel the development of skin lesions but seems to occur independently.

Microscopically, the neuritis of lepromatous leprosy is characterized by progressively increasing thickness of the perineurium, giving the appearance, in cross section, of an enlarged nerve. Because of the fibrosis the nerve becomes thick, stiff, and cordlike. Bacilli and vacuolated lepra cells also are prominent. (Later, I will further describe muscles involved through damage to the nerves.)

Lepra Reactions

The term is given to the transient bouts of fever that occur in the majority of patients with lepromatous leprosy, either with or without sulfone treatment.

These may vary from mild transient bouts of fever with some aggravation of existing skin lesions and the appearance of new lesions to extremely severe attacks characterized by high fever, aggravation of all existing lesions with inflammation and superficial necrosis, and the appearance of new lesions. Erythema nodosum is usually present. Many patients have severe neuralgia during these attacks—often accompanied by bacillemia—and the nerves seem to be considerably swollen and exquisitely tender. Paralysis or loss of sensation may advance rapidly during such periods. The duration of the attacks may be from a few days to several weeks; no one has yet been able to explain why they occur.

Lepromin Reaction

The response called the lepromin reaction, or Mitsuda reaction, is thought to be an expression of defensive hypersensitivity to the products or component parts of the leprosy bacillus. This skin test, similar to the tuberculin test for tuberculosis, was devised and reported by Kensuke Mitsuda in 1919. He used an intracutaneous injection of phenolized boiled suspension of ground-up lepromatous granulation tissue in physiological saline solution. Since lepromatous leprosy is thought to represent a non-reactive form of the disease, the Mitsuda reaction is negative in lepromatous leprosy. In the tuberculoid type, an inflammatory nodule appears at the injection site after two or three weeks and, in some instances, may ulcerate. It is thought that the reaction is a specific one to the nucleoprotein of the *Mycobacterium leprae*. The test material has never been standardized and is not available commercially. This test is not a specific diagnostic test for leprosy, as there are positive findings in many non-leprous persons. The negative lepromin reaction suggests a lack of capacity for defense against leprosy and is considered to be a bad sign if leprosy is present and the reaction has been positive previously. A positive reaction does not mean that a patient has leprosy, but, if leprosy is present a positive reaction is considered a good sign because it suggests that there is a resistance to the disease.

TUBERCULOID LEPROSY

Tuberculoid leprosy is generally thought to be the self-limited form of the disease; the host reacts defensively and sometimes successfully against the bacillus. The disease is restricted to one or more limited areas of the body and, in some instances, there is eventual destruction of the bacilli and eradication of the infection.

Skin Lesions

Bacilli are extremely rare in the skin of patients with tuberculoid leprosy, and the skin lesions may vary considerably in appearance, as they do in the lepromatous form of the disease. The maculae are flat and hypopigmented and, although fairly common, are much less numerous than those of lepromatous leprosy and are usually found in exposed portions. The lesions are more sharply defined and discrete, are frequently dry, and sometimes rough. Loss of sensation in these lesions is more common than it is in lepromatous leprosy. Usually, the lesions themselves will show absence of sweating. Characteristically, the microscopic picture of tuberculoid leprosy consists of non-caseating tubercles with epithelioid cells. There is usually an abundance of lymphocytes and, in general, a resemblance to sarcoid.

The tuberculoid plaques may vary from small discrete papules to sharply elevated, red, heavily infiltrated plaques, very large in circumference. In large lesions, it is quite common for the central area to be clear, giving the appearance of two or more concentric annular lesions. Clinically, the lesions may resemble sarcoid of the skin, but the hyperglobulinemia and the eye lesions usually found with sarcoid are generally absent in the tuberculoid form of leprosy. The eye lesions of leprosy are limited almost exclusively to the lepromatous form of the dis-

FIG. 5-A
Boy with tuberculoid leprosy.

FIG. 5-B
Positive reaction to the lepromin test.

case. One of the most constant and characteristic findings of the tuberculoid type of leprosy is the presence of anesthesia in the involved skin.

Lesions of the Mucous Membrane

In contrast with lepromatous leprosy, in the tuberculoid form of the disease the mucous membranes of the upper respiratory tract are rarely involved. As mentioned before, the eyes are also frequently spared and only during the tuberculoid reactions is one liable to find acid-fast bacilli in the mucous membranes of the nose.

Visceral Lesions

The amyloidosis seen so commonly in patients with advanced lepromatous leprosy is rather uncommon in those with the tuberculoid type and, as a general rule, the internal organs are not involved. The findings of hyperglobulinemia, positive serological tests for syphilis, and elevated sedimentation rate are also less commonly seen in the tuberculoid form of leprosy.

Eye Lesions

The primary involvement of the anterior chamber of the eye is not often seen in tuberculoid leprosy where the most common eye lesion is the result of paralysis of the muscles of the eyelids and loss of sensation to the cornea, culminating in damage to the cornea through exposure. Even this, however, is not so common as it is in lepromatous leprosy.

Nerve Lesions

The frequent involvement of the nerves in tuberculoid leprosy was the reason that the old terminology of neural or maculoanesthetic leprosy was so widespread. As previously mentioned, in tuberculoid leprosy there is an early inflammatory reaction. This vigorous defense against the bacilli in the nerves destroys the nerve itself. It is the reaction of the host that causes the damage to the nerves, and it is probable that the localization of the involvement to a specific area by this reaction may result in more severe involvement of the single nerve in this area.

The loss of nerve function may be only partial, beginning with the sympathetic nerves. This results in loss of function of the sudomotor, pilomotor, and vasomotor nerves, followed rapidly by involvement of the fibers carrying the

FIG. 6
Typical involvement, with central clearing of the lesion.

sensations of touch, heat, cold, and pain. As a general rule, deep-tendon reflexes and pressure and vibratory senses are not lost, except very late in the disease. The motor-nerve involvement, except in acute reactions, usually develops gradually so that muscular weakness and loss of muscle tone precede actual paralysis and muscular atrophy. It is not uncommon in tuberculoid leprosy to see someone with mild involvement of the extremity—as far as the motor paralysis is concerned—but with an extensive loss of sensation that far exceeds the involvement of the motor distribution of a given nerve.

A little-understood part of the nerve involvement in both types of leprosy are the trophic changes, characterized by concentric absorption of the phalanges and metatarsals, and changes of the Charcot type in the smaller and, occasionally, major joints. The widespread loss of sensation in the extremities is responsible for the high incidence of perforating ulcers on the extremities. This is especially common in the feet because of weight-bearing and other contributing causes. One of these is paralysis of the small muscles of the feet due to involvement of the tibial nerve, resulting in claw toes and calluses beneath the metatarsal heads. The absence of normal pain perception leads to the rapid development of ulcers in these anesthetic feet. The same is true of the hands, where burns, abrasions, cuts, and other injuries frequently go unnoticed until secondary infection appears.

In tuberculoid leprosy, with its nerve involvement, the nerve structure is still recognizable in early lesions, despite a heavy inflammatory infiltrate. In old and extremely thickened nerves, the architecture is obliterated by a sarcoid-like infiltrate of epithelioid-cell tubercles. Caseation necrosis, which sometimes leads to the formation of a nerve abscess, is seen rather often in nerve lesions, in contrast to skin lesions in which it is rather rare. In general, in tuberculoid leprosy bacilli are more easily discovered in nerve than in skin lesions.

In contrast to the lepromatous type of leprosy, the nerve involvement of tuberculoid leprosy is usually not symmetrical and the degree of nerve involvement may have no relationship to the extent of the generalized involvement. For example, in some patients who have had only one reaction there may be complete

TABLE V

DOSAGE SCHEDULES OF THE VARIOUS SULFONES

Drug	Method of Administration	Dose per Day (*Grams*)	Rest Period between Doses
DDS *	Oral	0.50 to 0.2	Variable
Diasone	Oral	0.3 to 1.0	1 week in 4, and 1 month in 4
Promin	Intravenous	2.0 to 5.0	1 week in 4, or 2 weeks in 12
Promizole	Oral	4.0 to 16.0	1 day in 7
Sulphetrone	Oral	1.0 to 3.0	Variable

* Parent sulfone (4, 4′ diaminodiphenylsulfone).

loss of motor and sensory function in an extremity, which is all out of proportion to the extent of the general involvement.

As in lepromatous leprosy, exacerbations or reactions may occur also in the tuberculoid form of the disease. In a tuberculoid reaction the lesions become inflamed, edematous, more elevated, and reddened. The involved nerves, which are often painful and hypersensitive, usually become acutely swollen and, occasionally, even abscessed. During a reaction, bacilli may be encountered in the tissues and, in some instances, a low-grade fever may manifest itself. If these reactions do not subside within the normal period of time—a few days or weeks—and if they recur repeatedly, it may indicate a conversion from the tuberculoid form of the disease to the lepromatous.

MEDICAL TREATMENT

The method of treatment in leprosy is so complicated and so varied that it has been a subject of social and medical literature since historical times. Before the use of sulfone, the most widely employed preparation was chaulmoogra (hydnocarpus) oil and its ethyl esters. There was no standard combination of these drugs or of the dosage; these varied as much as the method of introduction—orally, intradermally, subcutaneously, intramuscularly, or intravenously. Some doctors found that intensive chaulmoogra treatment was effective, but others found it to be almost valueless; in many leprosaria the use of chaulmoogra oil had been abandoned a decade or two prior to the use of sulfone.

The modern-day treatment of leprosy began in 1941 in the United States Public Health Service Hospital at Carville, Louisiana, where a sulfone called promin was first used. This substance—p,p′-diaminodiphenylsulfone-N,N′-didextrose sulfonate—had been originally developed for the treatment of tuberculosis. Because of the similarity between the organism causing tuberculosis and that causing leprosy, the drug was given a clinical trial. Within a year, it became apparent that it was more effective than anything that had been tried previously and that, even in patients with advanced lepromatous leprosy, the skin became bacteriologically negative within a period of two to five years. It must be added, however, that some advanced lesions never achieved bacteriological negativity. Promin is given intravenously in doses varying from one to five grams, according to the body weight.

Other derivatives of the parent substance, frequently known as DDS 4,4′ diaminodiphenylsulfone—diasone, sulphetrone, promizole, and promacetin—have been tried at Carville and are being used in other parts of the world. Oral administration is an advantage in diasone, which is widely used in Central America. Sulphetrone is most popular in India and Africa, but requires three times the dosage of diasone. Most workers feel that the parent substance is the most satisfactory of the sulfone derivatives. It has the advantage of being cheaper than some of the proprietary preparations. It must be pointed out, however, that because of the potentiality of a resultant anemia, the initial dose of any of the sulfones must

be small. The full dosage should be reached cautiously—by weekly, or even bi-weekly, increases.

Antibiotics have all been used but none seem to be as useful as the sulfone derivatives. Streptomycin and dihydrostreptomycin appear to be of some value as an adjunct in some patients, especially those sensitive to the sulfones. Aureomycin has also been reported to have been used successfully at Carville.

Because leprosy is a very slow-moving disease, except during reactions, response to treatment is likewise slow. Generally, the size of the lesions and the number of the bacilli in them are slowly reduced over a period of a year or more. Lesions of the eye and upper respiratory tract are usually arrested by treatment and may even regress slowly. Ulcers tend to heal more rapidly under treatment than they would if untreated.

Erickson reported relapses occurring after apparent arrest in patients undergoing treatment with sulfone. These occurred from one to three years after the start of treatment; the average time for the appearance of the relapse was forty-five months after the onset of treatment with promin. During the relapses, numerous old lesions reappeared and new ones developed. All the lesions were loaded with bacilli. Of the six patients with relapses, only one continued to receive sulfone, but under reduced dosage. The cause and ultimate outcome of these relapses is not yet known.

Side effects of the sulfone.. are not common but they may be serious. Anemia is an important reaction which may complicate sulfone therapy, and, occasionally, neutropenia may occur. These side effects usually respond to the administration of iron and cessation of the sulfone. Some workers have found that starting the treatment with smaller doses and then gradually increasing them has lessened the occurrence of these reactions. Other complications, such as allergic fever, erythema nodosum, dermatitis, and hepatitis, may also result from sulfone treatment.

Cortisone and its derivatives have been of value in treating some of the acute lepra reactions by diminishing the formation of fibrous tissue. This medication might possibly be of value in acute reactions by lessening the amount of swelling occurring in a nerve and preventing permanent nerve damage. However, there may occur other complicating factors that would contra-indicate the use of cortisone during acute reactions, but cortisone has not been used enough to know what they might be. The local use of cortisone or its derivatives in the treatment of eye lesions has been of great benefit; locally, the drug has none of the undesirable effects of systemic treatment.

One of the recent forms of therapy, still too new to be evaluated, is marianum antigen. This antigen was first used by Sister Marie-Suzanne of Lyon, France, who isolated and cultured a mycobacterium—called *Mycobacterium marianum* in her honor—from a leprous nodule. Intradermal monthly injections of a killed culture of these organisms were given to some volunteers in the treatment of leprosy. At Carville, a group of patients who failed to respond to prolonged sulfone treatment were selected for treatment with the marianum antigen, but it is too early to say whether the vaccine is beneficial. Some local reactions to the antigen have occurred, which may indicate some type of activity on the part of the vaccine. No biopsies of the reactive lesions have been studied at the time of this writing, and the antigen is being used only as a supplement to sulfone or other therapy.

Another form of therapy which is also new and cannot yet be evaluated is the use of bacille Calmette-Guérin vaccine in combination with drug treatment. Again, it is too early for any conclusions to be drawn concerning this type of therapy.

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The Hand in Leprosy

A Seven-Year Clinical Study

Part II. Orthopaedic Aspects of Leprosy *

BY DANIEL C. RIORDAN, M.D., NEW ORLEANS, LOUISIANA

To my knowledge, there has never been any comprehensive literature on the orthopaedic aspects of leprosy. There have been a number of communications over the years concerning the roentgenographic bone changes in leprosy, but these have not been correlated with orthopaedic treatment in any true sense of the word. The bone changes seen in leprosy in both the upper and the lower

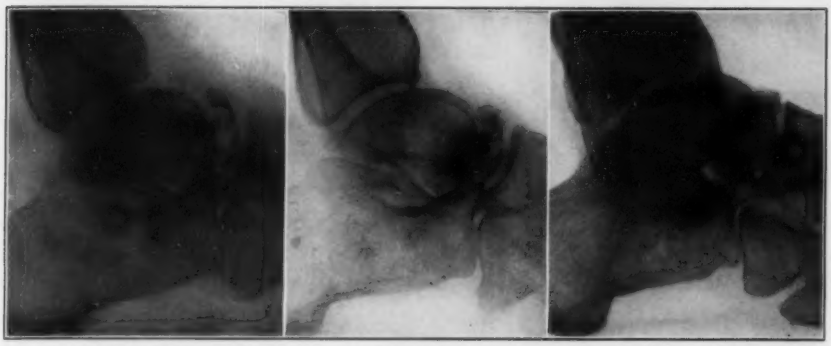

Fig. 1-A Fig. 1-B Fig. 1-C

Fig. 1-A: Leprous osteitis of the neck of the talus (roentgenogram reversed).
Fig. 1-B: Lesion healing under medical treatment and immobilization in a cast.
Fig. 1-C: Healed lesion.

Fig. 2-A

Figs. 2-A, 2-B, and 2-C: Bone absorption in the foot.

* Part III, dealing with reconstructive surgery of hand deformities in leprosy, will be published in a subsequent issue of The Journal.

FIG. 2-B

FIG. 2-C

extremities will be discussed here even though the prime purpose of this paper is to present my experience in the treatment of the upper extremity involved by leprosy.

In a general way, the changes seen in the bones and joints of patients with leprosy may be broken down into five entities: (1) leprous osteitis of the bone, thought to be a true leproma caused by the *Mycobacterium leprae;* (2) periostitis, considered to be due solely to the presence of the *Mycobacterium leprae,* without secondary infection; (3) absorption of bone in the upper and lower extremities, occurring in both major types of leprosy and thought to be due chiefly to the nerve involvement; (4) osteomyelitis, frequently superimposed on pre-existing bone changes of the types already mentioned, when ulcerations penetrate to underlying bone; and (5) destructive joint changes of leprosy (which I have grouped under the term Charcot joints), undoubtedly secondary to the nerve changes.

LEPROUS OSTEITIS

As early as 1889 Sawtschenko reported nodelike changes in the spongiosa

FIG. 3-A

Bone absorption in the hand.

of the bones in leprosy. He described these as being similar to the lesions in the skin and noted the presence of bacilli. He definitely distinguished between this type of bone involvement and the absorptive and inflammatory periostitis.

Hansen and Looft, in their textbook on leprosy, also described a leprous lesion in the bone marrow.

Faget and Mayoral described similar involvement, thought to be a true leproma, in the talus. This lesion was described as being a typical destructive lesion which had its onset during an acute lepra reaction, when bacilli are most numerous in the blood. It healed spontaneously without drainage under medical treatment and plaster-cast immobilization. This type of lesion can be identified only in roentgenograms of excellent quality, and care must be taken to rule out secondary infections or other causes.

PERIOSTITIS

Apparently the first description of roentgenological changes in the periosteum caused by leprosy was by de la Camp in 1900. He described subperiosteal new-bone formation as seen in roentgenograms and on pathological examination and stated that the changes were those of a chronic periostitis due to the leprosy bacillus in the periosteum and bone marrow. Since that time, many others have described periosteal changes. The changes may be due to direct involvement of the periosteum by bacilli or to superficial ulceration and secondary infection. In my experience no significant clinical findings have been associated with periostitis caused by *Mycobacterium leprae*, although some interesting roentgenograms have been seen.

BONE ABSORPTION

Much has been written concerning bone absorption due to leprosy and,

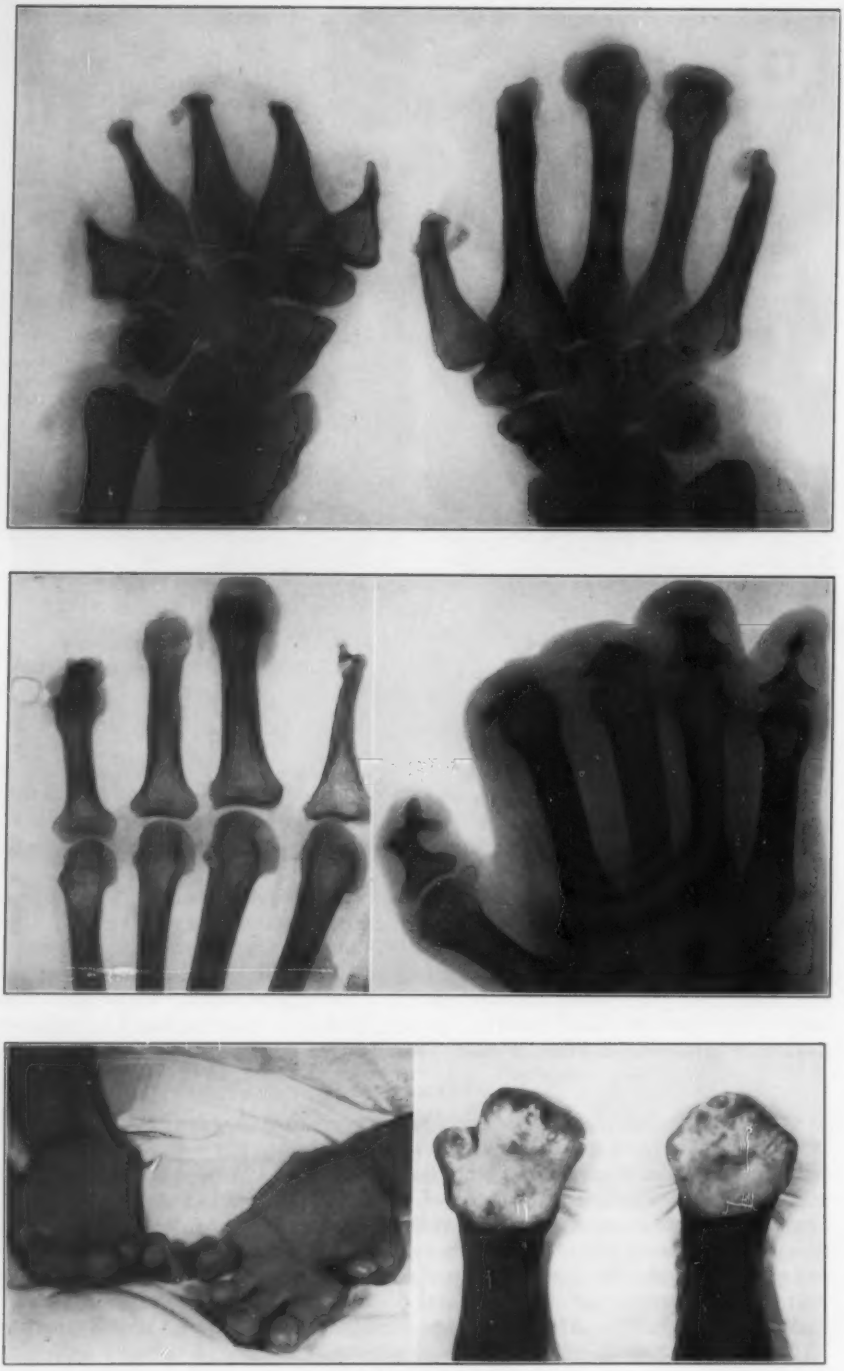

FIG. 3-B

Bone absorption in the hand; note fingernails on the stumps.

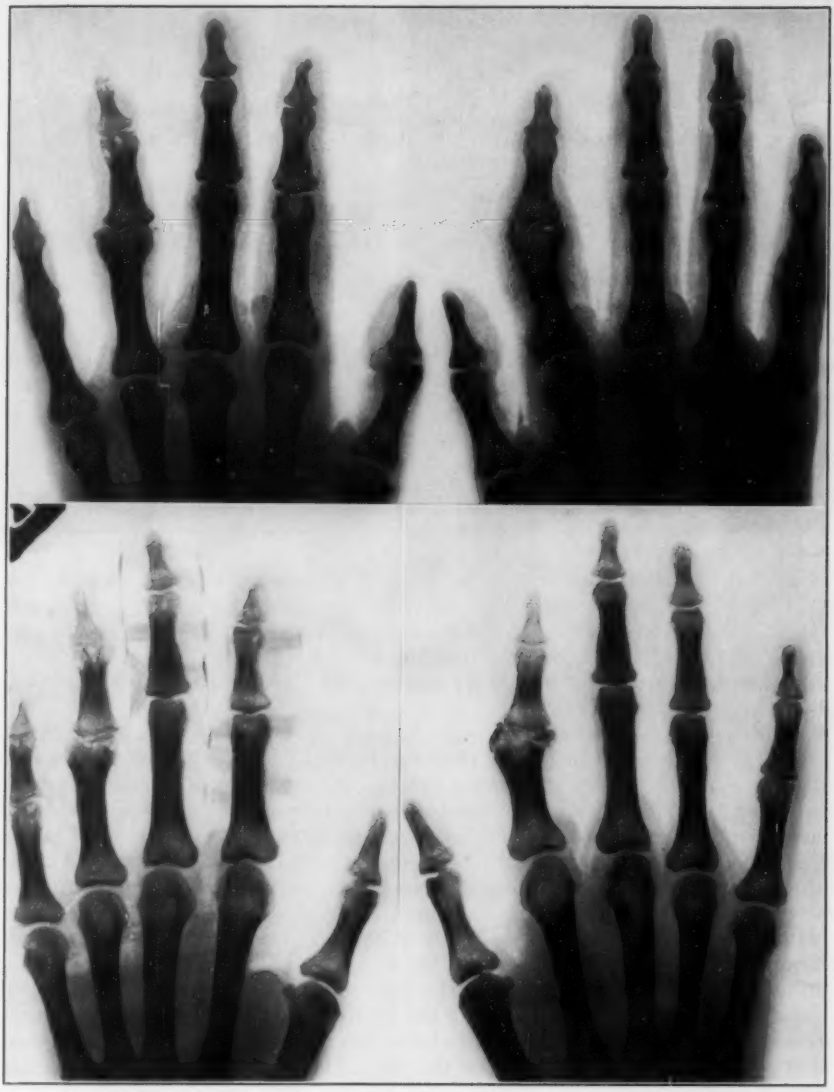

FIG. 4

Enlarged nutrient foramina and bone cysts with fracture.

although no one offers any satisfactory explanation for it, most authors agree that the absorption seen in the extremities is the result of the nerve changes and not of destruction of bone by the leprosy bacillus.

Kalindéro in 1897 believed that the absorption of the bone was caused by changes in the nerves, which affected the nutrition of the bones. He postulated that this produced osteomalacia, with resorption of the calcium salts, and bone atrophy. Most of the writers since the earliest reports on bone changes of leprosy pointed out that the bone-absorptive changes—the ones most frequently described—are associated with the nerve changes and therefore are more common in tuberculoid leprosy. These changes, however, are seen in the later stages of both

FIG. 5-A
Charcot ankle.

FIG. 5-B
Total absorption of the bones of the foot.

types of leprosy with almost equal frequency. Again and again, throughout the years, authors have pointed out that the absorption of bone is not caused by inflammation, atrophy of disuse, leprous osteomyelitis, or periostitis, but is the result of neurotrophic changes.

In the lower extremities the earliest change seen in bone absorption is thinning of the proximal phalanges of the toes. This may progress to the point where the bone gets so thin in the central portion of the proximal phalanx that it is easily fractured. It is not uncommon to see a metatarsophalangeal joint absorbed completely, leaving a toe which is fairly normal in appearance, but flail. Absorption of the metatarsal heads is quite common, and, next in frequency is complete absorption of the bones of the toes. This absorption may be so marked as to involve all of the phalanges and metatarsals, and, although extremely uncommon, complete absorption of all the bones of the foot has sometimes been observed. A popular misconception is that the digits of the feet or hands become necrotic and drop off as a result of leprosy. Actually, the bone undergoes absorption, and the soft tissues gradually shorten to adapt to the shortening of the supporting structure. It is not uncommon to see a patient able to flex and extend the soft-tissue stump that remains of either the toes or the foot. The fingernails and toenails may remain, but they undergo atrophy and become very small. The nail may be present even after complete absorption of the associated toe and metatarsal or the finger and metacarpal. Sloughing of digits occurs only in the presence of secondary infection and swelling, which interferes with the circulation and leads to necrosis. Erickson and Johansen, in 1948, pointed out that the bone changes of leprosy are slowed down under adequate therapy and that small bone cysts may actually heal under sulfone therapy. They also pointed out, however, that, where extensive neural involvement is present prior to treatment, secondary bone changes due to absorption may continue to increase despite sulfone treatment. They suggested that the disappearance of bone cysts is due to the healing of actual bone lepromata.

Another form of bone absorption is the cystlike formation found in the fingers and the enlargement of nutrient foramina of the bones in the digits. These small bone cysts and enlarged nutrient foramina have been described by a number

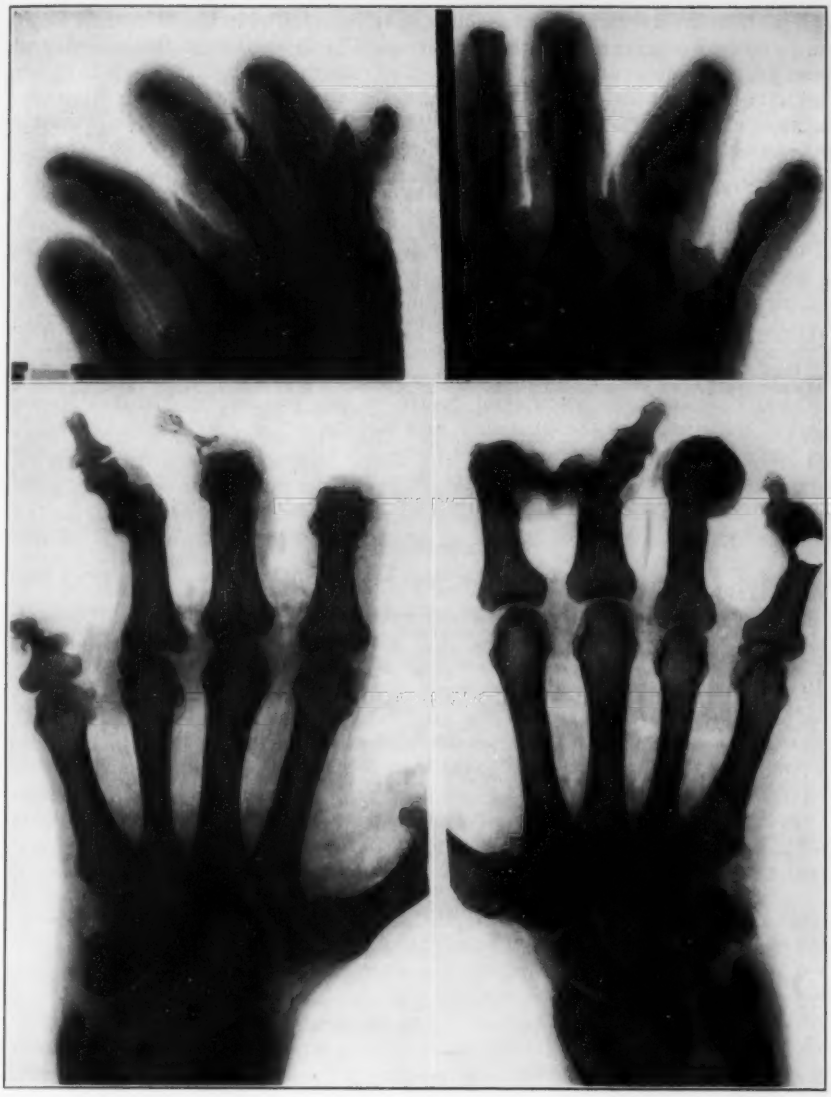

FIG. 6

Charcot fingers.

of authors. Most authorities agree that these are caused by actual bone lepromata, although their pathogenesis has not been proved.

CHARCOT JOINTS

Unstable joints resulting from neurotrophic involvement are seen in leprosy as well as in other diseases. Neurotrophic involvements in leprosy, however, differ from those in other diseases in that the majority of them are confined to the interphalangeal, metatarsophalangeal, and metacarpophalangeal joints. It is uncommon in leprosy to see involvement of a major joint, such as the ankle or wrist, and I have not observed it in the knee, hip, elbow, or shoulder. Furthermore,

no reports of involvement of these joints have been found in the literature. From the prolonged observation of these patients, it is thought that the majority of these unstable joints are purely the result of repeated minor traumata to joints that have no pain fibers to warn the patients that injuries have occurred. Minimum fractures of the margins of the joints at the ligamentous attachments are seen if roentgenograms are made whenever any swollen digit is discovered.

OSTEOMYELITIS

Osteomyelitis in leprosy is due to secondary infection which, in practically all cases, is indirectly the result of the nerve damage. Loss of sensation leads to ulcers, either from weight-bearing or from untreated injuries. The ulcers become deep and extend down to the underlying bone, providing entrance for secondary infection. This is usually readily detected by roentgenogram because of the appearance of sequestra and of periostitis; this periostitis is usually different from that caused by the *Mycobacterium leprae*. These changes are seen most frequently around the metatarsal heads or the metatarsophalangeal joints. Marked destruction of the phalanges and metatarsals may result; occasionally, the tarsal bones are destroyed when there are ulcers over these areas. In the fingers these secondary infections are usually confined to the interphalangeal joints and digits, although occasionally they involve the metacarpals.

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Acute Hematogenous Osteomyelitis

A Review of Sixty-six Cases

BY JACK L. WINTERS, M.D., AND IRVIN CAHEN, M.D., NEW ORLEANS, LOUISIANA

From the Department of Orthopaedic Surgery, Louisiana State University School of Medicine, New Orleans

Acute hematogenous osteomyelitis is a common and at times tragic disease from which severe deformity and disability may result. With the introduction of antibiotic therapy, a major improvement in the treatment of acute hematogenous osteomyelitis was obtained. Early in the antibiotic era the results of adequate therapy were most gratifying; however, recent experiences have indicated that this initial favorable response has not continued and that this disease no longer responds to antibiotic therapy in the same degree as it did when these drugs were first used. Although great advances have been made during the past two decades in the treatment of osteomyelitis, recent studies over the past four years have indicated that this disease is increasing in its incidence and in its morbidity. In addition, the disease appears to be changing in several of its accepted clinical characteristics.

Because of our observations that acute hematogenous osteomyelitis has altered its character, a critical analysis of all of the cases of this disease treated at Charity Hospital in New Orleans from January 1956 to and including the first four months of 1959 has been carried out. All patients with a diagnosis of acute hematogenous osteomyelitis admitted to Charity Hospital during this time interval are included in this study. All patients on whom follow-up was considered adequate were observed for a minimum of six months. Many of the other patients were studied for a year, and a considerable number have been followed for almost two years. This report has been completed in order to present certain aspects of clinical experience relative to this disease and has four aims:

1. To focus attention on the current relatively high incidence of acute hematogenous osteomyelitis in southern Louisiana and the apparent yearly increase in the over-all number of cases;

2. To discuss the modification of clinical symptoms which has occurred during the past decade and to suggest the possibility that this change may have resulted from the influence of antibiotics;

3. To offer basic proved methods of therapy;

4. To recommend surgical drainage in carefully selected cases as a procedure that may be indicated more frequently under the present concepts of this disease entity.

INCIDENCE

The total number of patients found in this survey was sixty-six—thirty-five male and thirty-one female patients. This almost equal sex distribution differs considerably from the six-to-one preponderance of males that has been cited in much of the earlier literature regarding this disease. In the past it has been assumed that trauma, to which males are more frequently subjected, accounted for the relatively high incidence of acute hematogenous osteomyelitis in that sex. Perhaps trauma, if it is an important factor, is not so limited to one sex in our present generation as it was in the past when females were more restricted physically and hence less frequently exposed to trauma. In addition, the disease appears to be becoming more frequent in infancy when trauma is less likely to be a significant factor.

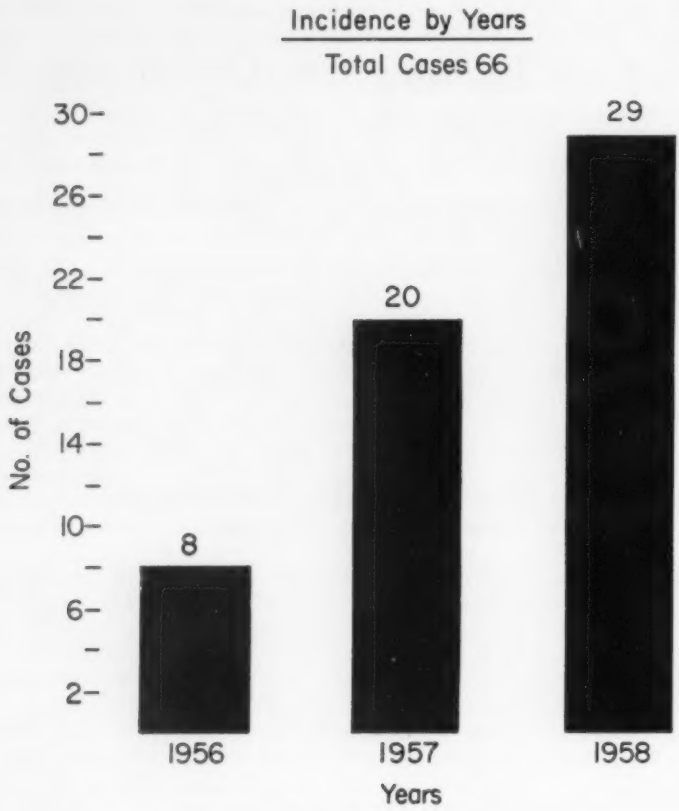

CHART I

During the years 1956, 1957, and 1958 the incidence of this disease increased greatly (Chart I). This increased number of cases of osteomyelitis occurred during a period in which the total number of admissions to Charity Hospital did not change significantly. Although our studies in 1959 are incomplete it would appear that this steady increase in incidence will continue. In the initial four months of 1959, nine cases were encountered.

In this series of sixty-six patients, all age groups were represented: the oldest patient was sixty-seven years of age, and the youngest was nine days old. Five were adults, and sixty-one were children (fifteen years of age or less). This observation is in keeping with the fact that acute hematogenous osteomyelitis most often involves rapidly growing bone. Of our five adult patients, four had significant co-existing disease. Three of these patients had diabetes mellitus and one had sickle-cell anemia. In childhood the absence of serious co-existing disease was significant.

Of the sixty-one cases occurring in childhood, sixteen (23 per cent of the entire series) occurred in children less than one year of age. Chart II graphically represents the age incidence of acute hematogenous osteomyelitis in this series.

ETIOLOGY

In the past it has been stated that *Staphylococcus aureus* is the etiological organism present in 80 per cent of the cases of acute hematogenous osteomyelitis. The remaining 20 per cent are said to be caused by *Streptococcus hemolyticus* and

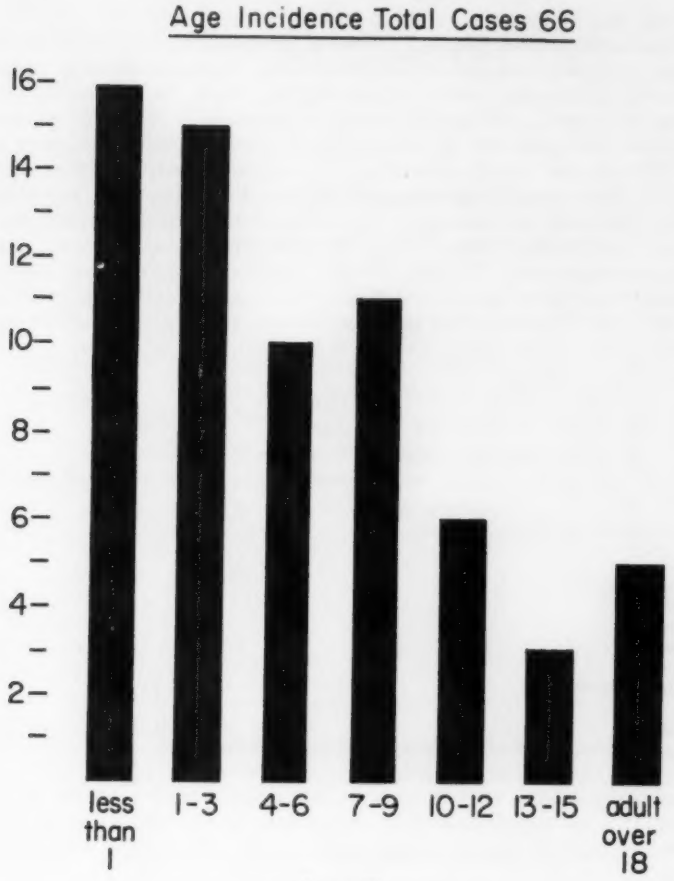

CHART II

Infants less than one year of age accounted for 23 per cent of the entire series.

Escherichia coli, with a variety of other organisms found occasionally. Moreover, it has also been stated that *Streptococcus hemolyticus* is the most commonly encountered organism associated with this disease in infancy. Our bacteriological studies confirm only some of these earlier findings.

The etiological organism may be cultured from two sources. Early in the disease blood cultures will often be positive. Later, purulent material may be aspirated locally from within soft-tissue abscesses or from the subperiosteal zone. In fifty-two of our sixty-six cases specimens were submitted from one or both sources for bacteriological study. In fourteen cases no specimens for culture were obtained, usually because of inaccuracy in early diagnosis. Early but inadequate antibiotic therapy often obscures the clinical symptoms, and diagnosis is then not made with certainty until typical features appear in the roentgenograms. These features include areas of radiolucency occurring usually in the metaphysis of a long bone, periosteal proliferation, and various degrees of sequestration and reactive sclerosis. Early in the disease deep swelling of the soft tissues with a loss of demonstrable muscle planes may be observed, but this roentgenographic sign is neither constant nor pathognomonic. In these instances blood cultures are of no value since they are invariably negative, and, usually, no purulent material can be obtained from the subperiosteal area.

Of the fifty-two specimens submitted for culture, only five revealed absence of bacteriological growth (Table I). Four of the patients from whom these sterile specimens were obtained had received antibiotic treatment prior to admission to the hospital. In the forty-seven remaining specimens, the etiological agent was identified. In forty-one of these the causative organism was *Staphylococcus aureus*. This represents 90 per cent of the specimens in which positive cultures were obtained. Salmonellae were isolated from two patients, both of whom had sickle-cell anemia. Paracolon, *Streptococcus hemolyticus*, *Proteus vulgaris*, and *Hemophilus influenzae* were each encountered once. In the sixteen patients under one year of age, *Staphylococcus aureus* was isolated from six of the eleven specimens submitted for bacteriological study. No cultures were obtained from the five remaining patients under one year of age. Four cultures were negative and one grew *Hemophilus influenzae*. It is of interest that streptococci were not identified as the etiological organism in any of the cases occurring in infancy.

TABLE I

BACTERIOLOGY

SIXTY-SIX TOTAL CASES — FIFTY-TWO CULTURES OBTAINED

Etiological Organism	No. of Cases	Percentage of Cultured Cases
No growth	5	9.6
Staphylococcus aureus	41	79.2
Salmonella	2	3.6
Paracolon	1	1.9
Streptococcus	1	1.9
Proteus vulgaris	1	1.9
Hemophilus influenzae	1	1.9
Total	52	100.0

Sensitivity studies were done in most instances when positive cultures were obtained. It is of significance that 57 per cent of the strains of *Staphylococcus aureus* encountered in this series and tested for penicillin sensitivity were sensitive to the drug.

BONES INVOLVED

In the sixty-six patients studied, there were seventy-two bones involved. In sixty-two patients the disease was confined to one bone—usually the metaphysis of a long bone. In four of the patients, bone involvement was multiple. Chart III is a schematic representation of the frequency with which the individual bones were involved. The region of the knee, which is the area most often subjected to trauma and the site of most rapid skeletal growth, was affected in over half of the cases.

CLINICAL MANIFESTATIONS

The clinical manifestations of acute hematogenous osteomyelitis have been said to consist in rapid onset associated with chills, high fever, toxemia, dehydration, tachycardia, and pain in the affected extremity. There is usually severe localized tenderness over the metaphysis of a long bone. Within recent years, however, it has become apparent that the various signs of severe toxicity and septicemia are not now occurring with the same frequency with which they were previously observed by other investigators. Indeed, twenty-two (33 per cent) of our sixty-six patients were admitted with temperatures of less than 100 degrees and with no other systemic manifestations. Two case reports will serve to illustrate the varied nature of the clinical manifestations of this disease.

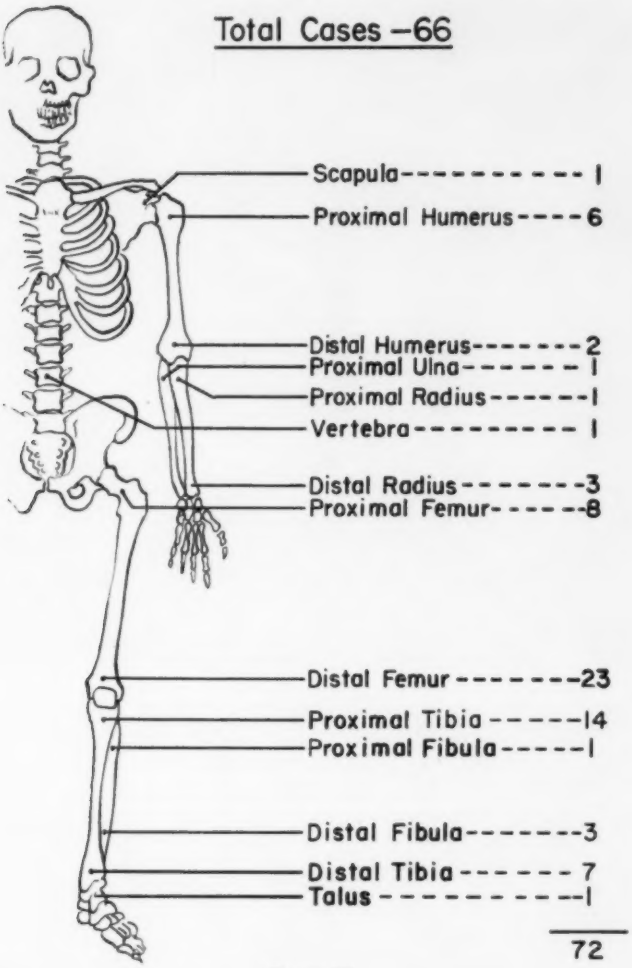

CHART III

Note that the region of the knee accounts for 51 per cent of the bones involved. In four patients multiple sites of infection were encountered.

A white male, J. S., three years of age, was admitted to Charity Hospital on February 3, 1959, with a history of a fall prior to the onset of illness. Several hours after injury chills and a high fever associated with severe pain in the region of the left knee joint developed. He was admitted to the hospital later the same day; at that time his temperature was 104.2 degrees. There were tachycardia and dehydration; point tenderness to palpation over the proximal metaphysis of the left tibia (no swelling or redness of the tissues was noted); and leukocytosis (30,000 leukocytes). Roentgenograms were normal (Fig. 1-A). A presumptive diagnosis of acute hematogenous osteomyelitis was made. Blood specimens were obtained for culture, and the left lower extremity was immobilized in a long plaster-of-Paris posterior splint with a pressure dressing. Aqueous penicillin, 300,000 units, and procaine penicillin, 300,000 units, intramuscularly along with chloramphenicol, 125 milligrams, orally were given immediately. Procaine penicillin, 300,000 units, was given every twelve hours, and chloramphenicol, 125 milligrams, orally was given every six hours. The patient remained febrile with temperature elevations of as high as 104 degrees for three days. Subsequently over the next five days, the temperature slowly declined, and he became afebrile. Blood cultures were returned on the third hospital day and revealed the presence of *Staphylococcus aureus*, coagulase positive, sensitive to penicillin and chloramphenicol. The extremity was inspected on the fourth hospital day and found to be less tender, but there was slight redness of the skin and mild warmth to palpation at the knee joint. A spica cast was thereafter applied and the antibiotics were

FIG. 1-A

J. S. Roentgenogram made on admission. Soft-tissue swelling was present, but there was no evidence of bone disease.

continued. Twenty days following admission the roentgenogram revealed a radiolucent area in the metaphysis of the left tibia (Fig. 1-B).

The patient was hospitalized for a total of thirty-four days during which time antibiotic therapy was continued and the affected limb was immobilized in a plaster spica cast. He was then dismissed from the hospital; at home he received additional antibiotic therapy for two weeks. During this time, erythromycin, 125 milligrams, four times a day was substituted for penicillin. Spica immobilization was continued for one month following hospital dismissal; a toe-to-groin cast was then applied and worn for an additional two months. Ten weeks after the onset of his illness, while he was still in a toe-to-groin cast, a pathological fracture occurred through the proximal end of the left tibia (Fig. 1-C). With continued immobilization, the fracture healed in excellent position. When the boy was last seen nine months after the onset of illness, only palpable thickening of the proximal end of the tibia and sclerosis, seen roentgenographically, served to identify the site of the infection (Fig. 1-D). At no time was there external drainage; the patient is now considered to be well.

The second case illustrates the more chronic and indolent type of the disease, which is being observed more and more frequently in recent years.

A Negro female, A. J., three years of age, was admitted to Charity Hospital January 12, 1959, with a history of having had several furuncles some three weeks prior to her admission. She was given penicillin for this furunculosis. Two weeks prior to admission, she complained of pain in her right leg, and a week later would not walk on the affected extremity. Upon admission she appeared well but had a slight limp, favoring the right lower extremity. The proximal portion of the right leg was swollen and warm and very tender. Some fluctuation was noted in this area. The patient was afebrile, and there was no leukocytosis. Aspiration of the fluctuant area was accomplished, and four cubic centimeters of yellow purulent material was obtained and submitted for culture. Cultures revealed the presence of an aerobic gram-positive rod assumed to be a contaminant. Penicillin, 300,000 units, was given every eight hours for nearly four weeks; during this time the limb was immobilized in a spica cast. She was dismissed from the hospital thirty-five days after admission. The cast was removed one month later; there was no external drainage. Two weeks after removal of the cast, pain and swelling of the involved area developed. On examination a soft-tissue abscess of the anterior proximal portion of the leg was noted. Aspiration was again done. Cultures revealed *Staphylococcus aureus*, resistant to penicillin and sensitive to chloramphenicol. The limb was im-

mobilized in a toe-to-groin cast for a month. The patient was given chloramphenicol, 125 milligrams, orally four times a day. After the cast was removed, two draining sinuses over the proximal portion of the tibia were observed. When the child was last seen, over six months after the onset of the disease, there was intermittent drainage from these sinuses. At that time the roentgenographic appearance seemed improved, and the child was active and had no obvious deformity. Figures 2-A through 2-C illustrate the course of the disease in this patient.

In this series one-third of the patients had a similar indolent form of acute hematogenous osteomyelitis.

TREATMENT

Treatment for most of these sixty-six patients with osteomyelitis was conducted by orthopaedic residents under the supervision of staff orthopaedic surgeons. Although the responsibility for treatment was delegated to many individuals, treatment was fairly uniform. Therapy consisted in three major factors: antibiotics, immobilization of the affected part, and surgical intervention when indicated. In addition general supportive measures, such as adequate fluid intake, bedrest, and blood transfusions were utilized when necessary.

Antibiotic therapy is the most important measure in the treatment of acute hematogenous osteomyelitis. Although all of the common antibiotics were used in the treatment of this group of patients, parenteral penicillin, in combination with other antibiotics, usually chloramphenicol or erythromycin, was given immediately upon admission. The most often used antibiotic regimen consisted in aqueous penicillin, 300,000 units, and procaine penicillin, 300,000 units, both given intramuscularly; with these, 500 milligrams of chloramphenicol or erythromycin was given orally immediately on admission after specimens were obtained for culture. Procaine penicillin was then continued in the dosage of 300,000 units intramuscularly every twelve hours and chloramphenicol or erythromycin was given orally at the rate of half a gram to a gram a day. Antibiotic dosage was very

FIG. 1-B

FIG. 1-C

Fig. 1-B: Roentgenogram made twenty days after admission. Radiolucent areas are now visible in the proximal metaphysis of the left tibia.

Fig. 1-C: Roentgenogram made ten weeks after admission. There is a pathological fracture with abundant callus formation in the proximal metaphysis of the left tibia.

FIG. 1-D

Roentgenogram made nine months after admission. The pathological fracture is healed. There is moderate sclerosis of the proximal tibial metaphysis.

rarely less than that indicated even in small infants; in older children and adults the dosage was increased accordingly. In addition, if purulent material could be aspirated from the site of involvement, antibiotics were instilled locally. Instillation of antibiotics was accomplished in approximately one-half of the patients. Usually 500,000 units of aqueous penicillin dissolved in five cubic centimeters of normal sterile saline solution was injected. When sensitivity studies were returned, the antibiotics were selected according to the results of these tests. No serious reactions to these antibiotics were encountered.

In recent years there has been a tendency, particularly among individuals not specifically trained in orthopaedic surgery, to neglect immobilization of the extremity; this is an integral part of the management of this disease. Immobilization of the affected extremity, preferably by a plaster cast, offers support to the extremity and serves to prevent deformity. By placing the affected part at rest, dissemination of the infection by means of venous or lymphatic routes is decreased by reducing muscular activity. Similarly, if periosteal perforation has occurred, dissemination of the infection through the soft tissues may likewise be diminished by immobilization. Moreover, certain complications are more apt to occur in patients treated without the benefit of immobilization. These include bone and joint deformity, pathological fracture, and dislocation of a septic joint. In this series of sixty-six patients, eleven were treated without early immobilization of the affected part. These patients were managed initially by the general hospital staff and were later treated on the orthopaedic service when immobilization was instituted. Of these eleven patients three had pathological fractures, one had

FIG. 2-A

A. J. Roentgenogram made on admission. A radiolucent area with a sclerotic periphery is noted which involves the anterior portion of the proximal metaphysis of the right tibia. Considerable periosteal new-bone formation is evident.

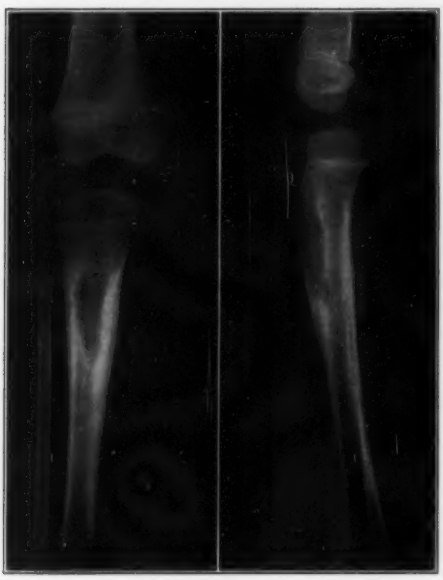

FIG. 2-B

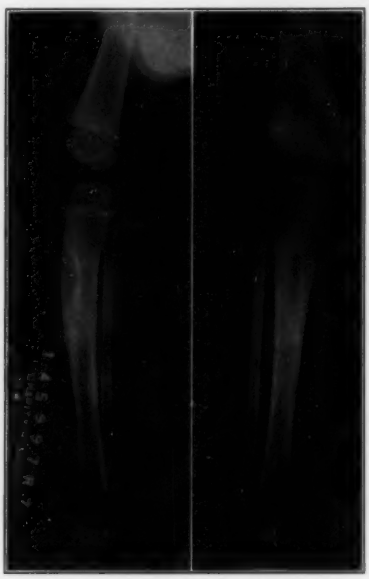

FIG. 2-C

Fig. 2-B: Roentgenogram made four weeks after onset of disease. There has been an increase both in the size of the lesion and in the amount of periosteal new-bone formation.

Fig. 2-C: Roentgenogram made three months after onset of disease. A marked diminution is seen in the size of the lesion, but a radiolucent area surrounding sclerosis is still evident.

dislocation of a septic hip, and two had chronic osteomyelitis as evidenced by persistent draining sinuses and the formation of sequestra. Of the five remaining patients one had an inadequate follow-up, and four had uneventful healing. The incidence of complications in this group of patients treated without immobiliza-

tion was 60 per cent, which is considerably higher than the incidence of unfavorable results in the series as a whole.

In this group of patients, fifty-six were treated conservatively—that is, without incision and drainage but with antibiotics, immobilization, and general supportive measures. Of the fifty-six patients, healing without significant residua occurred in thirty-eight. Chronic osteomyelitis developed in twelve; in these further treatment was required. Follow-up was considered to be inadequate in six patients. Acute osteomyelitis was classified as chronic when after a reasonable period of time resolution of the original infection had not occurred as evidenced by persistent sinus formation with drainage and the formation of obvious sequestra visible on roentgenograms. Continued prolonged drainage from an operative wound also was considered to put a case in the category of chronic osteomyelitis. Minimum to moderate persistent sclerosis as seen roentgenographically, with thickening of the involved cortical bone, in the absence of clinical symptoms was considered to represent healing of the infection. Of those patients treated conservatively and followed for an adequate period (six months or more) 76 per cent were considered to be completely well six months after the onset of the illness.

Although in recent years the role of surgery has assumed much less importance than in former years in the treatment of acute hematogenous osteomyelitis, surgical intervention is still necessary in carefully selected cases. Incision and drainage, with or without fenestration of the cortex, is the most frequently done and is probably the only operation indicated in acute hematogenous osteomyelitis. Surgical intervention becomes necessary if favorable response to the usual conservative measures does not occur. Indeed, when the systemic manifestations are not favorably influenced by antibiotics, immobilization, and other supportive measures, incision and drainage may become a life-saving procedure. This operation is always indicated when there is progression of the local infection even if the systemic manifestations of the disease are controlled by antibiotics. Incision and drainage was performed on ten patients in this series after they failed to respond to conservative therapy. Of these patients, two, although they improved initially after drainage, were not followed to an end result; in three healing was uneventful; and in five chronic osteomyelitis developed.

In review of the therapeutic measures utilized, it is obvious that the basic approach in the treatment of these patients has been conservative. It is our impression that the results in the group of patients operated on would have been improved by earlier surgical intervention. In the indolent variety of the disease, which is seen so frequently at the present time, and which so often becomes chronic (as evidenced by the fact that in 36 per cent of our patients admitted with temperatures of less than 100 degrees chronic osteomyelitis developed), one must observe the progression of the local lesion very closely, for despite the apparent benign course in this form of the disease, a progressive destructive bone infection not infrequently occurs. Perhaps incision and drainage may offer a better solution than conservative care in those patients who do show a lack of systemic manifestations and yet a slow indolent progression of the local lesion. We propose to treat some of these patients in the future by early incision and drainage.

COMPLICATIONS

The development of chronic osteomyelitis is the most common complication of acute hematogenous osteomyelitis. Seventeen of our patients who were observed for adequate periods of time were considered to have chronic osteomyelitis. In addition, pathological fracture occurred in four patients, in three of whom chronic osteomyelitis developed. Septic dislocation of the hip was seen in two patients. One child was followed for nearly three years and, at the time of writing, was three years old. No ossification center for the capital femoral epiphysis was present and the hip was dislocated. The proximal half of the femoral shaft was sclerotic, and there was one inch of shortening of the affected extremity.

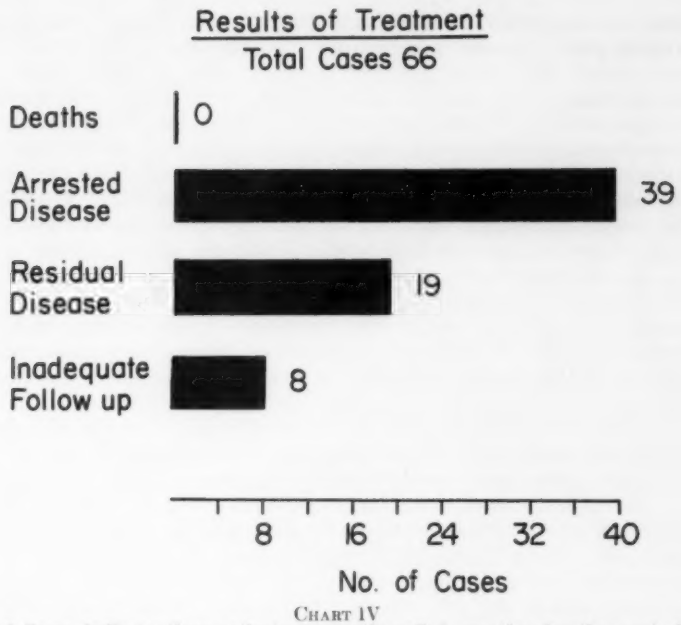

CHART IV

Arrested disease indicates those patients apparently well six months after the onset of disease. Residual disease denotes chronic osteomyelitis or dislocation of a septic joint. Inadequate follow-up includes those patients observed for less than six months.

The capital femoral epiphysis had probably been destroyed; a hip reconstruction is now being considered. The second patient was nearly two years old and had been observed for eighteen months at the time of writing. The hip was then not dislocated, but there was, as yet, no ossification center for the capital femoral epiphysis. The child was in a Frejka pillow splint and there was one-half inch of shortening of the involved extremity. No significant length inequality secondary to accelerated growth was observed. There were no deaths in this series of patients, and no other significant complications were recorded. Chart IV summarizes the results of treatment.

DISCUSSION

Acute hematogenous osteomyelitis is indeed a disease in which significant change has occurred and is now taking place. This change has been produced in great measure by two factors—the use of the antibiotics and the remarkable propensity of the *Staphylococcus aureus* to develop resistance to almost all antibiotics. With the advent of the widespread use of penicillin somewhat over a decade ago, it became apparent that the number of cases of acute hematogenous osteomyelitis seen in our hospitals was decreasing. Within recent years it appears from the study reported here that a rebound phenomenon is occurring and that acute hematogenous osteomyelitis is now increasing in its incidence. There are two causes for the increase in incidence of acute hematogenous osteomyelitis manifested in this series. First, the house staff at this hospital has certainly become more aware of the disease in recent years and the diagnosis is being made more accurately. Second, when the majority of the strains of *Staphylococcus aureus* were still sensitive to penicillin, many cases were probably completely aborted by early antibiotic treatment without hospital admission. Recently with the progressive increase in the number of bacteria resistant to the usual antibiotics, fewer cases are being aborted in their early stages. As a matter of fact, it is our distinct impression that early but inadequate antibiotic treatment may contribute

to an increased tendency toward the development of chronic osteomyelitis as well as making the diagnosis more difficult. Similar observations have been made by Bose.

The fact that sixteen cases (23 per cent) occurred in infants less than one year of age is probably significant. According to Blanche, in 1952, the incidence of acute hematogenous osteomyelitis was decreasing except in infants less than one year of age. Since the diagnosis is often an obscure and a difficult one in infancy, perhaps these young patients are not receiving early antibiotic treatment, and thus the disease in infants is not being aborted very frequently in its early stages. In this study a yearly increase in incidence during the last three years was found in all age groups, and this increase was most significant in infants.

In this series salmonella osteomyelitis occurred in two patients with sickle-cell anemia. Within recent years this association of diseases has been reported quite commonly. An excellent report is that of Silver, Simon, and Clement in 1957. Other authors including Hughes and Carroll, Hook and associates, and Sanquinette, have reported the co-existence of these two diseases.

In the years preceding the widespread use of antibiotics, *Streptococcus hemolyticus* was frequently reported as being the most common offending organism causing acute hematogenous osteomyelitis in infancy. Such observations were made by Green and Shannon in 1936, by Dickson in 1945, and by Compere, Schnute, and Cattel in 1945. During the last three years it has been reported by Wolman (1956) and by Clarke (1958) that *Staphylococcus aureus* is the most common cause of acute hematogenous osteomyelitis in infancy. As a matter of fact, both of these authors did not report a single case in which the *Streptococcus hemolyticus* was the etiological agent. In our own cases in infants less than one year of age, *Staphylococcus aureus* was the only etiological agent which could be identified except for one infection caused by *Hemophilus influenzae*. It is our belief that *Streptococcus hemolyticus*, which is characteristically quite sensitive to penicillin, has been practically eliminated by antibiotics as a causative organism in acute hematogenous osteomyelitis in infancy.

That one-third of our patients were admitted with temperatures of less than 100 degrees and with no systemic manifestations is a modification in the clinical manifestations of acute hematogenous osteomyelitis. Why this disease should be associated with less systemic reaction now than it was in the past is not clear. Two factors may be implicated. First, many of the patients with this disease received antibiotics prior to admission to the hospital. This antibiotic therapy, which is often given in inadequate dosages on the basis of a faulty diagnosis, apparently aborts the systemic manifestations of the disease leaving a local smoldering infection which continues within the bone of the affected extremity. Second, a significant number of patients within this series (10 or 15 per cent) were less than six months of age, when fever is not a prominent manifestation of infection. Indeed, nine of our sixteen patients who were less than one year of age were afebrile on admission to the hospital. Wolman stated that the most striking feature about acute hematogenous osteomyelitis in infancy is the generally good condition of the infant with little or no fever or signs of toxicity. Clarke made similar observations in 1958.

Altemeir and Reinecke, in 1945, said, "Penicillin has revolutionized the management of acute hematogenous osteomyelitis. The spectacular control of the bacteremia, the bony infection, and the metastatic visceral complications has produced a radical reduction in morbidity as well as mortality". Unfortunately, such enthusiasm is no longer warranted. In this series of cases, 43 per cent of the strains of *Staphylococcus aureus* encountered were found to be resistant to penicillin. Clarke in 1958 found no sensitive strains at all in his cases.

It is quite true that the mortality rate associated with acute hematogenous osteomyelitis is infinitely smaller than the 10 to 15 per cent mortality rate reported by Dickson in the preantibiotic years. On the other hand, how different

the disease is now compared with the time when penicillin was a novel drug. In 1945 Agerholm and Trueta reported thirty patients with no deaths and twenty-eight excellent results and also in 1945 Compere, Schnute, and Cattel reported twelve patients treated with penicillin with no deaths and excellent results in all of them.

In 1947 Higgins, Browne, and Bodian reported thirty-one cases with no deaths and no other complications. In the series reported here, in one patient in every four, despite antibiotics, immobilization, and surgery when indicated, chronic osteomyelitis developed as evidenced by continued drainage and sinus formation or obvious sequestration as shown by the roentgenogram.

It is now obvious that acute hematogenous osteomyelitis has changed during the antibiotic era and has reached a stage where the casual use of antibiotics will no longer bring about a cure. In order to treat this disease successfully, the physican must employ every means at his disposal. These include, not only the use of antibiotics, but also the sound principles of immobilization and early adequate incision and drainage in those patients in whom surgery is indicated. The physician must not be guilty of treating patients with prolonged antibiotic therapy while the destruction of bones and joints continues. He must not be reassured by the indolent course and lack of systemic abnormality which may mask a smoldering progressive infection within the involved bone. Incision and drainage is indicated and should be resorted to whenever the local lesion continues to progress despite conservative treatment, even if the patient is afebrile through this clinical phase.

SUMMARY

A review of all the patients with acute hematogenous osteomyelitis admitted to Charity Hospital during the period January 1, 1956, to May 1, 1959, was undertaken to verify the clinical impression that this disease is increasing in frequency and changing in character. Sixty-six patients were seen during this time and are included in this study. A significant increase in the incidence of acute hematogenous osteomyelitis has occurred in southern Louisiana over the past four years. This increase in incidence has been ascribed to the increasing number of bacteria resistant to the usual antibiotics and particularly to the resistance of *Staphylococcus aureus* to penicillin.

In contrast to previous reports the sex distribution in the series was almost equal. Twenty-three per cent of the patients were one year of age or less.

Staphylococcus aureus was identified in 90 per cent of the patients in whom positive cultures were obtained. *Streptococcus hemolyticus*, contrary to many early reports, was not identified as the etiological organism in any of the infants.

One-third of our patients were admitted with a very mild form of this disease and had no significant fever or other systemic manifestations.

Treatment consisted in antibiotics, immobilization, and surgery when indicated. Despite what was considered adequate treatment in almost every instance, chronic osteomyelitis developed in 29 per cent of the patients in this study who were adequately followed. It is our impression that early but inadequate antibiotic treatment caused an increased tendency to the development of chronic osteomyelitis.

Additional emphasis should be placed upon the basic orthopaedic principle of immobilization. In this survey complications developed in the majority of the patients who were not treated initially by immobilization.

Antibiotics have unquestionably decreased both the mortality and morbidity associated with acute hematogenous osteomyelitis. Nevertheless, as more bacteria become resistant to our common antibotics, the incidence of chronic osteomyelitis will continue to increase. Perhaps more liberal utilization of early incision and drainage in those cases which are prone to become chronic, as evidenced by slow indolent progression of the local lesion, may improve the present results.

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Traumatic Dislocation of the Hip Joint in Children

A REPORT BY THE SCIENTIFIC RESEARCH COMMITTEE
OF THE PENNSYLVANIA ORTHOPEDIC SOCIETY *

Traumatic dislocation of the hips is an uncommonly seen lesion in the child, and the available information about its behavior on which to base a prognosis is meager. The literature contains, for the most part, scattered case reports with conflicting opinions as to end results and complications.

Nicoll, in 1952, reported the results of a survey by a clinical research group of The British Orthopaedic Association investigating the results of traumatic dislocation of the hip joint. They found the incidence of aseptic necrosis of the femoral head to be 10 per cent in the adult. They were unable to present any statistics in children but they believed that, theoretically, the incidence in children might be greater. Blount, on the other hand, stated that aseptic necrosis is a less common sequel of dislocation of the hip in children than in adults but that when it does occur the end result is more devastating. Blount was also of the opinion that aseptic necrosis is unlikely to occur in a child if there is no evidence of necrosis three months after the reduction. Banks reviewed all the cases of aseptic necrosis in children after traumatic dislocation of the hip reported up to 1941. He concluded that aseptic necrosis in children who were up to about ten years of age appeared similar to Perthes' disease and responded favorably to non-weight-bearing. In older children, on the other hand, the changes after aseptic necrosis resembled those seen in adults, with equally poor results.

The last comprehensive review of the literature concerning this subject was done by Mason and covered the period from 1922 to 1954. He was able to find eighty-eight reported cases of traumatic dislocation of the hip in children. The incidence of aseptic necrosis in these cases was 10 per cent, which was the same incidence reported by Nicoll in adults. Of 193 dislocations of the hip reported by Stewart and Milford, only seven occurred in children. They found the incidence of aseptic necrosis to be 15.5 per cent in the hips reduced by closed methods, and 40 per cent in those reduced surgically. They believed that the age of the patient does not materially affect the end result. In 1959 Cros Juan reported one case with a follow-up of three and one-half years. The total number of cases of traumatic dislocation of the hip in children reported up to 1959, therefore, is ninety-six.

It is evident from the available historical data that no investigator to date has studied a sufficient number of these cases to predict with any degree of authority the behavior of a dislocated hip or its prognosis after traumatic dislocation in children. The Pennsylvania Orthopedic Society set out in 1959 to collect and analyze the cases of traumatic dislocation of the hip in children treated by its membership. This study is to continue over a five-year period and has as its ultimate aim the answers to the following questions:

1. What is the incidence of aseptic necrosis in children after traumatic dislocation of the hip?
2. What factors, if any, influence the incidence of aseptic necrosis?
3. What is the end result of a traumatic dislocation of the hip in a child at the time of bone maturity?
4. Does a traumatic dislocation of the hip in a child differ in any way from a similar lesion in the adult?

* Read at the Annual Meeting of the Pennsylvania Orthopedic Society, Pittsburgh, Pennsylvania, October 23, 1959.

It is hoped that this study will aid in dispelling the existing uncertainty about this lesion by the cataloguing of a significant number of these cases and the observation of their behavior over many years of follow-up study.

MECHANICS OF STUDY

In 1957 the Pennsylvania Orthopedic Society formed The Scientific Research Committee. The function of this committee is to initiate studies at the State level on chosen orthopaedic topics that lend themselves to statistical evaluation on the basis of collected questionnaire information. The Committee acts as a group to collect, evaluate, and disseminate information received from its membership on these topics. In 1958 the Committee reported on an evaluation of the treatment of fresh mid-shaft fractures of the humerus in adults in Pennsylvania from 1952 through 1956[7].

The topic chosen for study in 1959 was traumatic dislocation of the hip in children. A total of thirty-two previously unreported cases was collected and studied to date (October 1959). It was decided by the Society to continue this study over the next five-year period in order to answer accurately the four questions posed previously. During this five-year period accurate follow-up data on the present group of cases will continue to be collected. All new cases occurring during this period will be catalogued and followed.

A preliminary report based on these thirty-two cases is presented now, and a final comprehensive report will be made at the completion of the five-year study period.

FINDINGS

Of these thirty-two dislocations, sixteen occurred on the right and sixteen on the left side. Twenty-five (78 per cent) were in males, and seven in females. The ages of the patients at the time of diagnosis ranged from five weeks to fifteen years. There were two age periods when dislocation of the hip appeared to be more frequent, with 34 per cent occurring between the ages of four and seven years, and 41 per cent occurring between the ages of eleven and fifteen years.

Information regarding the mechanism of injury was not specifically requested in the original questionnaire, but this aspect will be thoroughly explored at the time of the final report. The cause of the dislocation is known, however, in sixteen of the thirty-two cases. Eight occurred in automobile accidents, five in falls of various types, one while the patient was playing baseball, one followed a parental spanking, and one occurred at birth following a breech delivery. Information made available to the Committee concerning the dislocated hip in the newborn indicated that this represented a true dislocation rather than a separation of the epiphysis with metaphyseal fracture. Complete information is being sought from the attending surgeon.

There were six anterior, twenty-four posterior, and two central dislocations. In twenty-two patients dislocation of the hip was the only lesion; in five patients there were associated fractures of the acetabulum; and in the remaining five patients there were associated fractures of the femoral head, neck, and greater trochanter. No open dislocations or fractures occurred, and there was no involvement of the sciatic nerve in any of the patients. Absence of all arterial pulsations distal to the hip was observed in association with one anterior dislocation due to impingement of the head of the femur on the femoral artery. Normal pulsations were restored after immediate closed reduction. In four instances the dislocation went unrecognized for five weeks, four months, five months, and six months, respectively. The results to date in these four cases will be given in detail later in this report. One patient suffered a second traumatic dislocation of the same hip one year after the first. The result in this case was rated *excellent* six months after the second dislocation.

As shown in Table I, the majority of the dislocations was reduced without

TABLE I

TIME LAPSE BETWEEN INJURY AND REDUCTION

Dislocation Recognized		Dislocation Not Recognized	
Hours of Time Lapse	No. of Patients	Months of Time Lapse	No. of Patients
4 or less	15	1¼	1
12 or less	6	4	1
24 or less	5	5	1
48	1	6	1
96	1		

undue delay. Twenty-six of the thirty-two dislocations (81 per cent) were reduced within twenty-four hours after injury. It was shown in adults that a correlation exists between the incidence of aseptic necrosis and delay in reduction[8]. It is not accurately known if such a correlation exists in children. We hope to be able to furnish data on that point as we continue to catalogue and study these cases over the next five-year period.

Of the thirty-two dislocations, twenty-six (81 per cent) were treated by closed reduction, and two (6 per cent) by open reduction. The four remaining dislocations (13 per cent) were reduced by different forms of traction.

Table II shows in detail the types of immobilization employed for these patients. Interestingly enough, only 44 per cent of these patients were placed in a plaster spica immediately after reduction, whereas in 56 per cent some form of traction was used for immobilization either before or after reduction.

TABLE II

TYPE OF IMMOBILIZATION

Type of Immobilization	No. of Patients
Spica, immediately	14 (44 per cent)
Traction preliminary to reduction and spica	
Buck's (closed reduction)	4
Russell's (open reduction)	1
Skeletal (open reduction, screws in acetabulum)	2
Buck's traction only	4
Traction after reduction and before spica	
Buck's	5
Bryant's	1
Well-leg traction	1

When the dislocation was the only lesion, the period of non-weight-bearing averaged eleven weeks, and the period of partial weight-bearing averaged seven weeks. In dislocations complicated by fracture, however, the period of non-weight-bearing averaged fourteen and one-half weeks, and the period of partial weight-bearing averaged fourteen weeks.

The follow-up periods on all thirty-two patients ranged from three months to 167 months. In eight patients, who had been followed for six months or less, the lesions were considered too recent to evaluate, and three patients were lost to further follow-up. This left twenty-four patients available for evaluation.

The result was rated *excellent* when there was no pain, limp, or restriction of activity and when the hip joint appeared normal to clinical and roentgenographic examination at the time of evaluation. There were sixteen patients with adequate follow-up periods and no injury other than the dislocation. Excellent results were found in 81 per cent of these. Eight of the patients with dislocations complicated by fractures of the acetabulum or femur had adequate follow-up periods. Excellent

end results were recorded in 50 per cent of these. We regard these figures as tentative at best. It is our belief that an accurate evaluation of the end result after a traumatic dislocation of the hip in a child cannot be made until skeletal maturity is reached.

Seven patients (29 per cent) had results not rated *excellent* to date. Of these seven patients, five had dislocations associated with fractures of the acetabulum or the proximal portion of the femur, or both. In two patients the dislocation went unrecognized for four months and six months, respectively. In only one of these seven patients was the dislocation the only lesion present.

As shown in Table III the residual signs and symptoms found in this follow-up study were, in their order of frequency, limited hip-joint motion, limp, pain, and shortening of the extremity. Five of the twenty-four patients had some deformity of the femoral head, as seen on roentgenograms.

TABLE III

END RESULTS IN TWENTY-FOUR PATIENTS FOLLOWED FOR SIX MONTHS OR MORE

End Result	No. of Patients	Percentage
Limited hip motion	5	16
Residual pain	3	9
Residual limp	4	13
Femoral-head deformity	5	16
Residual shortening (1 inch or more)	3	9
Aseptic necrosis	2	6.3
(One patient seen at 1½ years after closed reduction)		
(One patient seen when diagnosis of dislocation was made 6 months after injury)		
Second operation	0	0

There have been two definite instances of aseptic necrosis to date in this series, and a third is strongly suspected. The first was noticed one and one-half years after closed reduction. The second instance of aseptic necrosis was present when the diagnosis of a previously unrecognized dislocation was made six months after injury. In regard to the patient with suspected aseptic necrosis, by the time of writing there was an interval of eight months since open reduction of the dislocation and fixation of an acetabular fracture with screws. Changes in the femoral head have occurred, but they are not yet definite enough to be classified as being due to true aseptic necrosis. As of October 1959, none of the patients in this series has required a second operation (Table III).

RESULTS IN DISLOCATIONS NOT RECOGNIZED AT THE TIME OF INJURY

CASE 1. The patient was a girl delivered by breech presentation on January 12, 1959. Traumatic dislocation of the left hip was diagnosed five weeks after birth and was thought to be the result of a birth injury. The attending surgeon believed that the lesion represented a true dislocation and not a slipped capital epiphysis. Reduction was effected by Buck's traction, followed by immobilization in a plaster spica. The hip was apparently normal when the infant was last seen at seven months of age.

CASE 2. The patient was an eleven-year-old girl at the time of injury in an automobile accident. She sustained a posterior dislocation of the left hip accompanied by a chip fracture of the femoral head and a fracture of the posterior rim of the acetabulum. Her original care was not under the direction of a member of the Pennsylvania Orthopedic Society. The hip was said to have been reduced by manipulation eighteen hours after injury but not immobilized. Roentgenograms seven weeks after injury showed the presence of dislocation. She came under the care of the reporting surgeon four months after injury. The dislocation was reduced by skeletal traction, followed by open reduction. Nine years after operation the patient was found to have limited motion of the hip, an antalgic limp, and one inch or more of shortening of the leg, and a short femoral neck and a flat deformed femoral head, as seen on roentgenograms. It is believed that this patient's disability is already so severe that a second reconstructive operation will be necessary.

CASE 3. The patient was a five-year-old girl with a deformity of he right lower extremity secondary to poliomyelitis. The child injured the left lower extremity in a fall and was placed under the care of a chiropractor. Posterior dislocation of the left hip was not recognized until the child was seen by an orthopaedic surgeon five months after injury. Treatment consisted in Buck's traction for one week, followed by closed reduction and immobilization in a plaster spica. At the time of this report, four months after the reduction, the child was still in the plaster spica.

CASE 4. The patient was a six-year-old boy at the time of injury. He sustained an anterior dislocation of the left hip and a long-bone fracture of the left leg. The fracture was recognized and treated, but the dislocation went unrecognized for six months. When the diagnosis of dislocation of the hip was made, aseptic necrosis of the femoral head was present. The dislocation was reduced by Jones' well-leg traction combined with adductor tenotomy. No weight-bearing was permitted for twelve months. At the time of follow-up six years after reduction, the hip appeared normal clinically. Roentgenographic examination revealed a coxa magna deformity and a reduced joint space.

COMMENT

1. A total of thirty-two cases of traumatic dislocation of the hip in children has been collected from orthopaedic surgeons in the State of Pennsylvania.

2. The majority of these cases was posterior dislocation in male children, with the greatest incidence in children in the age brackets of from four to seven and from eleven to fifteen years.

3. Traumatic dislocation of the hip was most commonly the only lesion.

4. Eighty-one per cent of the patients in this series were treated by closed reduction within twenty-four hours of injury.

5. When there was only traumatic dislocation of the hip, the period of non-weight-bearing and partial weight-bearing averaged eighteen weeks. In dislocation complicated by fracture, the period of non-weight-bearing and partial weight-bearing averaged twenty-eight and one-half weeks.

6. Seventy-five per cent of the patients had a follow-up period of six months or more.

7. When there was no injury other than the dislocation, an excellent result was obtained in 81 per cent of the patients in this series followed to October 1959. When the dislocation was complicated by fracture of the acetabulum or femur, an excellent end result was recorded in 50 per cent of the patients. We believe, however, that the true picture of the results after this injury cannot be obtained until skeletal maturity is reached.

8. The residual signs seen in the follow-up period, in their order of frequency, were limited hip-joint motion, limp, pain, and shortening of the extremity.

9. The incidence of proved aseptic necrosis in this series up to October 1959 was 6.3 per cent.

10. This study is to continue over the next five-year period, and: (a) all new data obtained and the results of all future follow-up examinations made of the thirty-two cases reported here will be filed with The Scientific Research Committee; (b) all new cases treated by members of this Society in the next five-year period will be recorded with The Scientific Research Committee; and (c) at the end of the five-year study period all the collected information will be evaluated, and a final report will be made.

Albert B. Ferguson, Jr., M.D., Pittsburgh (1958);
William F. Donaldson, M.D., Pittsburgh (1959);
Marvin C. Goodman, M.D., Lancaster;
Ervin E. Rodriguez, M.D., Lewistown;
Michael Skovron, M.D., Erie;
John J. Gartland, M.D., *Chairman*, Philadelphia.

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Genetic Implications in Coxa Plana

BY ALLAN INGLIS, M.D., NEW YORK, N. Y.

From the Hospital for Special Surgery, New York

Throughout medical science today there is increasing emphasis upon understanding the familial and associated genetic interplay in various disease processes. Studies of twins are of great value especially when the twins are identical, since each twin provides an ideal control for the other. There have been numerous reports in the literature of the familial incidence of Legg-Perthes disease. Despite the fact that there are many well studied pedigrees, frequently through several generations, there are few reports in twins. The following case reports are of interest because they present coxa plana in identical twins and, further, the incidence and mode of expression in identical mirror twins (Fig. 1).

FIG. 1

Both P. H. (left) and C. H. (right) on December 20, 1959, were six and one-half years old and had just learned to write. P. H. was allowed limited weight-bearing on the right lower extremity and C. H. had just been admitted to the hospital with advanced Legg-Perthes disease of the left hip.

CASE 1. P. H. was first seen in the Out-Patient Department and then was admitted to the Hospital for Special Surgery on April 16, 1957, at the age of four and one-half years. The mother had noted that he complained of vague pain in his right hip for about one month; further, that he had had a mild limp which had been becoming progressively more pronounced over this period. There had been no antecedent trauma and he had been well in every other respect. The family history revealed that he had a twin brother who was well; however, it had not been recognized that he was an identical twin.

At the time of admission the patient appeared well developed and of normal stature for a child of his age. He had a distinctly coxalgic gait and the right lower extremity was held in slight external rotation when walking. There was about 5 degrees restriction of internal rotation and of abduction with the hip flexed. The patient was right-handed.

The admission hemogram and urinalysis were within normal limits. The sedimentation rate was 10 millimeters in an hour. Roentgenograms revealed only the most minimal changes. The right capital femoral epiphysis was slightly smaller and denser than the one on the left. The opinion that this was Legg-Perthes disease was so strong that treatment was instituted. Following a brief period in traction, the patient was placed recumbent in a one and one-half plaster spica.

Roentgenograms made two months later substantiated the diagnosis, for there was mild early

FIG. 2-A

P. H. Roentgenogram made on June 18, 1957, two months after the diagnosis had been made and treatment had been instituted. The capital femoral epiphysis of the right hip is slightly smaller, flattened, and shows areas of early necrosis. The left hip is normal.

flattening and scattered areas of necrosis throughout the right capital femoral epiphysis (Fig. 2-A). During the ensuing twelve months more of the capital femoral epiphysis became necrotic and was resorbed (Fig. 2-B). Partial weight-bearing was allowed after nineteen months at the age of six when reconstitution of subchondral bone was deemed sufficient. The patient still has about 5 degrees restriction of internal rotation and a slight limp late in the day. Roentgenograms showed slight flattening of the capital femoral epiphysis and slight widening of the neck (Fig. 2-C).

FIG. 2-B

Roentgenogram made on March 3, 1958, twelve months after the diagnosis had been established. Most of the necrotic bone in the right capital epiphysis had been absorbed and there were early signs of early reossification.

FIG. 2-C

Roentgenogram made on August 11, 1959, twenty-eight months after the diagnosis of Legg-Perthes disease had been established. The patient had been fully ambulatory for nine months. This lateral view shows excellent reossification, especially in the subchondral areas. There is residual flattening of the capital epiphysis on the right with widening of the femoral neck.

CASE 2. C. H. The patient, the identical twin of P.H., was first seen in the Out-Patient Department of the Hospital for Special Surgery on August 15, 1958, at the age of five and one-half years because of pain in the left hip and a mild limp. He revealed no limp during the examination but he lacked about 10 degrees of internal rotation. Roentgenograms revealed no bone abnormalities (Fig. 3-A). It was appreciated at that time that the patient had a twin brother with Legg-Perthes disease. The patient was placed on bedrest at home and told to return in two weeks for re-evaluation. The pain and limp quickly subsided with a short period of bedrest and the mother felt that there was no need to keep the return appointment.

He was next seen a year later on December 1, 1959, at the age of six and one-half years because of pain and limp on the left during the preceding two weeks. Again, he was found to have mild restriction of motion of the left hip, but roentgenograms now demonstrated advanced changes in the left capital femoral epiphysis with flattening, areas of increased density, and other areas of bone resorption (Fig. 3-B). He was admitted subsequently.

The complete physical examination was unremarkable except for the left hip which was found to have 10 degrees restriction of both internal rotation and abduction with the hip flexed. It was also noted that he was left-handed. The hemogram and urinalysis were within normal limits. The sedimentation rate was 14 millimeters in one hour. He was subsequently placed in a duraluminum splint immobilizing both hips and was kept recumbent.

DISCUSSION

Further evaluation of the twins demonstrated that they were virtually mirror images of each other (Fig. 1). Subsequently it was noted not only that the handedness was asymmetrical as well as the hair whorl, but facial characteristics and fingerprint classification were also reversed. No two fingerprints are identical, of course; however, they may be classified much the same as blood type only with far more accuracy, with each finger having its own characteristics. In these children the right index finger of one was the mirror image of the left index of the other, and so on. Skin texture, hair texture and color, and iris color were all the same. Skin-grafting with cross transfers was not performed.

According to Newman, two-thirds of identical twins are symmetrical, that is, both right-handed, and one-third are asymmetrical or mirror images of each other, that is, one right-handed and the other left-handed. Minor mirroring or asymmetry includes opposite handedness, reversed hair whorl, fingerprints, dentition, facial characteristics, and birthmarks. Major mirroring occurs with *situs inversus viscerum* as well as the characteristics just mentioned. This is rare because anything less than complete expression of this leaves the cardiopulmonary or digestive

FIG. 3-A

C. H. Roentgenogram made on August 15, 1958, when the patient was five years old. Multiple roentgenograms of the left hip were all reported as normal; however, the patient complained of pain and limped on the left side.

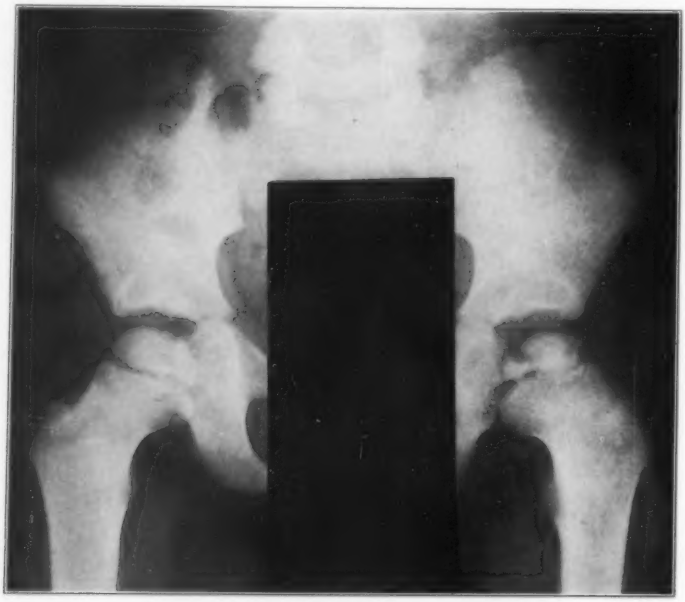

FIG. 3-B

Roentgenogram made on December 15, 1959, when the patient was six and a half years old and one year after initial symptoms and his visit to the Out-Patient Department. The left capital femoral epiphysis now shows moderately advanced necrosis and resorption with mild flattening of the epiphysis and widening of the femoral neck.

systems, or both, with major defects. These defects tend to prevent normal development and the fetus usually does not survive. The result is that only one twin survives and hence it is not appreciated that initially there may have been another twin.

The fact that P. H. and C. H. did not manifest their disease simultaneously is interesting. It is generally conceded that in early stages of growth one twin usually displays a slower rate of development than the other. This is presumed to have been caused by unequal division of placental nutrition. Most often this discrepancy in development diminishes as maturation continues.

Derian reported Legg-Perthes disease in both members of dizygotic twins. (Dizygotic twins are fraternal and have a different genetic constitution from each other. Genetically they are no more alike than any brother and sister.) One of the twins was symptomatic and the other was not when the diagnosis was made. Goff reported Legg-Perthes disease in one of the members of dizygotic twins. There are only two other reports in the literature of coxa plana in identical twins. One by Giannestras who reported the disease in twin brothers and also in their younger sister; and the other by Dunn, who reported it in monozygotic male twins. Both Derian and Giannestras emphasized the value of careful study of other siblings in a family in which coxa plana has occurred.

In a pedigree study, Stephens and Kerby reported the incidence of Legg-Perthes disease in a family through five generations. They found the disease appeared in twenty-eight of sixty-three children of parents, one of whom manifested the disease. They concluded that the genetic mechanism was a simple dominant type (Aa, aa), with variable penetration. Wansbrough and associates reviewed 130 cases over a thirty-year period. They found the family incidence to be about one in twenty-five while the incidence in the general population to be about one in twenty thousand. They concluded that this represented "incomplete dominance".

CONCLUSION

The familial tendency of Legg-Perthes disease has been well documented by the family incidence and even more strongly by an extensive study of family pedigrees. The twins studied by Giannestras and by Dunn and this report of the disease which is present in mirror fashion in identical twins illustrate even more vividly the genetic interaction in the disease. Therefore, those who are aware that coxa plana has appeared in a family should keep in mind the probability of the disease developing in another member.

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Proceedings

THE ORTHOPAEDIC RESEARCH SOCIETY

As Prepared by Dr. Paul H. Curtiss, Jr., *Secretary-Treasurer*, with the Assistance of the Contributors and Dr. Jonathan Cohen

The Sixth Annual Meeting of the Orthopaedic Research Society was held in Chicago, Illinois, January 22 and 23, 1960. The Officers for 1960 are as follows:

President: Ignacio V. Ponseti
President-Elect: Robert A. Robinson
Secretary-Treasurer: Paul H. Curtiss, Jr.

The Orthopaedic Research Society welcomes all who may be interested in applying for membership. The requirements for membership are primarily a present and continuing interest in research related to the field of orthopaedic surgery. The Society is particularly desirous of receiving applications from younger investigators, as well as from Canadian and other orthopaedic surgeons who feel that they will be able to meet the responsibilities of continuing membership. At the present time, membership is automatically forfeited by an active member if he fails to attend three consecutive meetings without suitable excuse, and, if for five consecutive years, fails to submit to the Program Committee a subject for presentation without suitable excuse.

Applications for membership should be sent to the Secretary, Dr. Paul H. Curtiss, Jr., 2065 Adelbert Road, Cleveland 6, Ohio.

The following new members were elected:

Ernest S. Barash
William J. L. Felts
J. William Fielding
Harold M. Frost
Paul R. Harrington
Lloyd A. Hurley
Floyd H. Jergesen
Leroy Stanley Lavine
Donald L. Stilwell, Jr.
C. R. Sullivan
Edgar Anthony Tonna
James Bailey Wray
* Richard B. Raney
* Frederic W. Rhinelander
* Frederick R. Thompson

* Denotes Senior Member

The next meeting of the Orthopaedic Research Society will be held immediately preceding the 1961 annual meeting of The American Academy of Orthopaedic Surgeons in Miami, Florida.

Application forms for submission of abstracts for presentation at the next meeting may be obtained from the Secretary. However, no abstract received after October 1, 1960, can be considered for the 1961 meeting. Abstracts received prior to August 15, 1960, will receive some priority in consideration for presentation at the Combined Meeting of the Orthopaedic Research Society and The American Academy of Orthopaedic Surgeons. One need not be a member of the Society to submit an abstract.

The papers presented at the Scientific Sessions are as follows:

Studies of the Lymphatic Pathways of Bone and Bone Marrow*. Dr. David W. Anderson [1] observed that the presence of lymphatics in bone and bone marrow has not been adequately demonstrated.

The work reported represented an attempt to confirm the findings reported by Oshkaderov, who introduced India ink into the medullary cavity of animals and then traced the course of the dye as it traversed the bone to the periosteum where it could be seen distributed throughout a fine reticulate mesh of channels which communicated with channels to the lymph nodes. He noted that within the bone cortex, lymphatic channels have the form of tortuous chains which are directed along Haversian canals.

The method used and reported in this paper was as follows: A portion of the medullary contents of the tibiae of rabbits was curetted and packed with gauze which remained in the tibia

1. 1118 Ninth Avenue, Seattle 1, Washington.

for seven days. This pack was then replaced with a gauze pledget soaked with India ink or other dye and the bone defect sealed with bone wax. The animals were sacrificed at intervals thereafter and histological sections prepared after gross dissections were completed.

The present study does not demonstrate the existence of lymphatic channels as an anatomical unit, but it does show what appears to be the flow of lymph from within the medullary space of bone through the Haversian canals.

* This study was supported by a grant from the Virginia Mason Foundation, Seattle, Washington.

The Role of Osseous Tissues in Bone Formation (A Preliminary Report) *. Dr. C. Andrew L. Bassett [2], Dr. Daniel K. Creighton, Jr., and Dr. Frank E. Stinchfield reported on a study utilizing a technique for producing isolated chambers within cortical bone. In one group of animals, defects, four by twenty millimeters, in adult, canine radii were segregated from both periosteal and endosteal elements by the use of either semi-permeable millipore filter or impermeable silicone rubber (silastic) membranes. After thirty days, abundant quantities of reactive new bone were found in the defects. Osteogenesis was always more advanced in the proximal ends of the gaps than in the distal ends. Bone formation seemed, in part, to result from migration of residual, endosteal cells and, in part, from cells arising in transected Haversian-canal systems. Healing was more rapid within cortical chambers with millipore walls than in those with silastic walls. Cartilage and osteochondroid matrix were found only in defects isolated within the impermeable silastic membranes. In a second group of animals, the cortical defects were exposed on their endosteal surface while the periosteal surface was covered by a wall of either millipore or silastic. These defects were obliterated more rapidly than those in the first group. Healing resulted primarily from a centripetal proliferation of endosteal callus, and did not differ significantly from that observed in control animals in which defects were exposed on both the endosteal and periosteal surfaces. Periosteal new bone was scanty in all animals.

* Supported in part by the Easter Seal Research Foundation, United States Public Health Service Grant A-1859, and a grant from the Squibb Institute for Medical Research.

Observations Which Pertain to an Alteration in Vitamin A Metabolism in Osteogenesis Imperfecta. Dr. Edwin G. Bovill, Jr.[3], reported that variations in configuration of the vitamin-A molecule are associated with variation in its potency. Variation in vitamin-A potency can produce either pathological fracture (hypervitaminosis A) or middle-ear changes similar to otosclerosis (hypovitaminosis A) in experimental animals. In some species of fresh-water fish a variation in molecular configuration of vitamin A (vitamin A_2) results in the formation of a purple retinal pigment (porphyropsin) rather than the orange pigment found in salt-water fish and human beings (rhodopsin).

These three observations warrant the opinion that vitamin-A metabolism may be the site of the metabolic disturbance responsible for osteogenesis imperfecta. To put this concept to a preliminary test a hypothesis was formulated which would relate the cardinal biological defects in this disease to a variation in the configuration of the vitamin-A molecule.

Data obtained from absorption spectrography in the ultraviolet range on the sera of normal subjects and of patients with osteogenesis imperfecta were presented. These data suggested that a significant variation in the molecular configuration of vitamin A does exist between the two groups studied.

Autoradiograms of the Femoral Head. Dr. R. A. Calandruccio [4] reported on his studies with radioactive phosphorus of femoral-head specimens which had been removed surgically from patients with (1) non-union of the neck of the femur, (2) avascular necrosis of the head of the femur, (3) fresh fractures of the neck of the femur, and (4) degenerative arthritis of the femoral head. These patients were injected with tracer doses of radioactive phosphorus approximately one hour before removal of the femoral heads. Autoradiograms of these specimens were obtained and were compared with the roentgenograms, gross specimens, and microscopic sections.

This material revealed the following: (1) P_{32} is not deposited in the avascular bone; (2) there is a sharp demarcation between the vascular and non-vascular tissue; (3) roentgenographic criteria are not adequate in determining viability of the femoral head; and (4) increased density of the femoral head may be produced by new bone superimposed on existing trabeculae.

Motor Unit Action Potential Frequencies in Human Muscle. Dr. J. R. Close [5], Dr. F. N. Todd, and Dr. E. D. Nickel stated that the normal electromyogram consists of a series of recorded action potentials produced by motor units firing at various frequencies. The number of

2. Columbia University, College of Physicians and Surgeons, New York, N. Y.
3. 450 Sutter Street, San Francisco 8, California.
4. 869 Madison Avenue, Memphis 3, Tennessee.
5. 2929 Summit Street, Oakland 9, California.

motor units recorded is influenced by the central nervous system through recruitment and variation of frequency. The resulting tension of contracting muscle thus depends upon the total number of action potentials produced. Total or analog counts of action potentials represent values related to the muscle's tension. Such counts are readily obtainable using electronic counters.

Tension was recorded through the use of bone pins inserted through the calcaneus and the tibial tuberosity to which the measuring devices were attached. The resulting tension curve during a contraction was very similar to the so-called analog action potential count rate curve.

Frequencies of action potentials of muscles throughout the body were found to vary, just as the sizes of motor units vary. Also, different frequencies and different counting thresholds were found in abnormal muscle.

The principal advantage of the method is that a continuous curve representing motor unit activity in quantitative terms is made possible.

A Description of Sarcomata Produced Experimentally Using the S. E. Polyoma Virus. Dr. Robert B. Duthie [6] said that the development of sarcoma in skeletal cells is determined by factors which are either chromosomal or extrachromosomal in origin, and these were reviewed briefly. By injecting the S. E. polyoma virus of Stewart and Eddy into golden hamster weanlings, many sarcomatous tumors arose within twenty-six to forty-two days in the heart, liver, kidney, and subcutaneous sites. Particular study was made of the development of malignant fibroblastic cells in the subepicardial area of the heart. Their histological appearance was described in detail with their reactions to the periodic acid-Schiff test and metachromatic staining techniques.

These primary sarcomata were found to be readily transplantable to both the cheek pouch and subcutaneous sites of homologous hamsters without previous conditioning of the recipient by x-irradiation or cortisone therapy. The desoxyribonucleic-acid content (by the Feulgren reaction) of the nuclei of these tumor cells was increased in amount and their uptake of radioactive sulphur (by autoradiography) was demonstrated.

Attempts were made to obtain survival of the animals with sarcomata by excising the primary tumor in the host; however, metastasis to the lungs occurred, and this behavior pattern was described.

Symposium on the Status of Plastic Polymer Investigation. Dr. Joseph E. Salvatore [7] and Dr. William Moncrief presented studies of implantations in animals of polyurethane polymer. Tissue cultures of HeLa cells were exposed to small particles of this substance. The cells showed slightly retarded growth during the first twenty-four hours, but normal growth for the next ten days. Implantation of pieces of polymer intraperitoneally and subcutaneously in rats and dogs resulted in marked diminution of the volume of plastic in some cases and in others the total dissolution of the plastic. The degree of tissue reaction was described as minimum but included cellular infiltrations and adhesions. Similar experiments in thirty rats over a period of twelve months and thirty-two dogs during the same period revealed similar tissue reactions locally but no systemic effects. One fibrosarcoma occurred after sixteen months in one of the rats. Another malignant tumor, apparently unrelated, occurred in a rat with involvement of the breast tissue.

Dr. Thomas J. Canty [8] then presented a summary of experimental observations on many different plastic substances, including hydrolyzed gelatin, plastic resins, and plastics composed of glycogen, glycine, calcium, and phosphorus. The methods of testing the materials physically and biologically were summarized and no specific claims were made concerning the plastic substances used. This presentation was one indicating the scope and methods of investigation but not attempting to delineate results.

Dr. Thomas L. Jones [9] and Dr. William M. King then presented a review of the concept of ostamer, a polyurethane plastic identical with that described by Dr. Salvatore and Dr. Moncrief. This study reported no acute toxic results after injection of finely ground suspensions of the plastic in carboxymethylcellulose administered subcutaneously into eighteen rats. Continuing studies on dogs over a period of three years also indicated no systemic sequelae but indicated that the plastic material showed signs of being absorbed and a variable degree of local tissue reaction. For the most part these authors described this reaction as minimum to *nil* both in the dogs followed for this period of time and in the rats treated similarly. Fractures immobilized by this substance were also studied and it was stated that repair bone could permeate the material with ease. The techniques of using the material were emphasized and clinical results were briefly alluded to. Since the Academy meeting to follow would include a more extensive review of the clinical studies, the discussion was confined to the experimental aspects.

Dr. Jonathan Cohen [10] stated that all of the preparations he was able to examine demonstrated significant tissue reaction of a degree which probably would prohibit the safe use of this material clinically.

6. University of Rochester School of Medicine and Dentistry, Rochester 20, New York.
7. Medical Corps, United States Army, Memphis, Tennessee.
8. Medical Corps, United States Navy, Oakland, California.
9. Cincinnati, Ohio.
10. 300 Longwood Avenue, Boston 15, Massachusetts.

DR. LENT JOHNSON [11] concurred in this opinion and reviewed the subject of fracture healing in the presence of this material. He stated that examination of many specimens seemed to him to indicate that this material delayed fracture healing rather than accelerated it.

DR. BERNARD BLOCH [12] pointed out that experimental advances sometimes had to be worked out despite apparent obstacles to the clinical application of the methods being tried. He stated that his animal experiments with other plastic materials in fracture healing were promising.

Some Studies Concerning the Epiphyseal Growth Zone Using the Modified Harris Test. DR. J. WILLIAM HILLMAN [13] and DR. JOHN A. BARROW, III, reported that the test devised by Harris for testing the tensile properties of the upper tibial epiphysis had been modified and extended. Standardization of the test was accomplished with definition of the limits of applicability. A variety of test situations was employed, particularly the response of the epiphysis to trauma, estrogenic and androgenic substances, cortisone, dietary alterations, and intravenous papain. In this presentation particular attention was given to the data obtained in the studies with papain and dietary rickets, which indicated that this test is capable of demonstrating early responses of the epiphyseal plate before gross or histological changes appear.

An example of the applicability of this test technique was demonstrated in the rickets experiment where the test indicated a failure of complete restoration of tensile strength in the rachitic animals when vitamin D was added to the diet. The semiquantitative biological test makes possible a more careful partitioning of the role of nutritional elements in bone growth and metabolism than is possible by the gross and histological appearance of the bone.

The administration of intravenous papain and the resultant mobilization of chondroitin sulphate from the epiphyseal-cartilage matrix were studied. The data obtained in these studies were confirmatory in some instances and paradoxical in others, in accordance with current knowledge of this plant enzyme.

Several clinical implications regarding growing bone were discussed.

Skin Arthroplasty of the Hip in Dogs. DR. NORMAN W. HOOVER [14] and DR. MARK B. COVENTRY observed that autogenous skin as an interposing material in arthroplasty had received only cursory notice until the past decade, whereas the deep implantation of skin for other purposes was well documented. The object of this study was to determine the fate of skin after its implantation within the joint of a dog, and its role in the formation of a new articular surface. Fifty-three arthroplasties were done upon the hips of thirty mongrel dogs, interposing fresh autogenous skin. Grafts of three types were used: full-thickness skin, the dermal layer, or the epidermal layer. The animals were observed for periods of one week to twenty-four months.

The skin was well tolerated, and infection occurred in only one instance. Skin used as an interposed membrane in arthroplasty of the dog's hip, regardless of the portion of skin used or its method of implantation, was found to be inadequate to withstand the intra-articular stress and did not survive as a weight-bearing membrane. A fibrocartilaginous joint surface developed, but there was no evidence of its origin from any portion of the implanted skin. Conversely, it seemed to stem directly from connective tissue of the intertrabecular spaces. An identical fibrocartilaginous surface was formed in the absence of implanted skin.

A Microchemical Study of the Developing Epiphyseal Plate. DR. ROBERT E. KUHLMAN [15] reported studies of the biochemistry of normal enchondral-bone formation in the dog by use of adaptations of established quantitative microchemical techniques. Five enzymes important to carbohydrate metabolism were measured at four stages of enchondral-bone formation. Enzymes mediating carbohydrate metabolism, not only through the Embden-Meyerhoff pathway and citric-acid cycle but also through the direct oxidative pathway, were found to be active in the portions of the epiphyseal plate composed of maturing cartilage cells. With the proliferation and subsequent hypertrophy of the cartilage cell, lactic dehydrogenase and phosphoglucoisomerase increased to peak activity and then decreased in the primary spongiosa area. On the other hand, glucose-6-phosphate dehydrogenase and malic dehydrogenase were most active in the primary spongiosa. Alkaline-phosphatase activity increased to reach a maximum in the primary spongiosa. In general, enzymes mediating metabolism through aerobic pathways were more prominent in the areas nearest to the sites of invasion by blood vessels, whereas those enzymes more important to aerobic glycolysis were more active in the avascular areas.

The total solid, the acid-soluble solid, and the phosphorus content at selected stages of bone formation were also investigated. Consideration of the enzymatically inert solids contained in the plate indicated that the epiphyseal area contains levels of enzyme activity comparable to those present in the cellular areas of brain, retina, and cornea. The quantity and variety of enzymes found in the epiphyseal plate indicate that enchondral-bone development is a process deriving energy and synthetic intermediates from an enzymatically regulated metabolic sequence. Methods

11. Armed Forces Institute of Pathology, Washington 25, D. C.
12. 149 Macquarie Street, Sydney, Australia.
13. Vanderbilt University Hospital, Nashville 5, Tennessee.
14. Mayo Clinic, Rochester, Minnesota.
15. Barnes Hospital, St. Louis 10, Missouri.

were presented for biochemical study of biopsy specimens from diseased epiphyseal plates.

An Experimental Attempt to Stimulate Growth by a Distracting Force across the Lower Femoral Epiphysis. Dr. H. O. Marsh [16], Dr. Erdogen Adas, and Dr. Kewal Laroia reported on experiments done in an attempt to stimulate longitudinal growth in the femur by a strong distracting force across the lower femoral epiphyseal line. Weaned Hampshire pigs were used. These animals have the necessary marked bone-growth potential at this age. Approximately twenty animals were used in getting the experiment started, testing the apparatus, and, finally, running the experiment. This report summarized the work on nine experimental animals and three controls.

Luschka's Joints: Degenerative Phenomenon. Dr. Caesar F. Orofino [17], Dr. Mary Sherman, and Dr. Daniel Schecter reported on their histological investigations of cervical spines of various ages, starting with spines from the prenatal period of development. Sections were made in both the coronal and the oblique planes. A thorough study of these sections and a comparison with a coronal section of an adult cervical spine leads them to the conclusion that no synovial joint exists in the region of the uncinate processes of the cervical vertebrae, and that the fissuring and fibrocartilage seen in that region along with the osteophyte formation are the result of a degenerative process.

Immunological Mechanisms of Bone Homografts. Dr. Henry H. Sherk [18] reported on experiments in which ten adult female grey Dutch rabbits were injected intraperitoneally with extracts of cortical bone from the New Zealand strain of white rabbits. The injections were given three times at six-week intervals and consisted of finely powdered bone suspended in saline solution and combined with Freund's adjuvant. Each animal was bled at a rate of ten cubic centimeters a week after the second injection. The sera of the test animals were evaluated for agglutination, precipitation, and complement-fixation antibodies against the extracts of donor bone.

Three weeks after the third injection, a cortical-bone graft from a New Zealand white rabbit was placed into the tibia of the corresponding grey Dutch host rabbit. The host response to the graft was evaluated histologically and compared with that in a control animal which had not been pretreated with injections of the bone extract.

The results of these studies seemed to indicate that there was an increased inflammatory response with a delay in reactive-bone formation about the bone grafts in hosts which had been pretreated with intraperitoneal injections of extracts of donor bone. There was no clear-cut evidence for the presence of a circulating antibody against these extracts.

16. The Wichita Clinic, Wichita 8, Kansas.
17. Ochsner Clinic, New Orleans 15, Louisiana.
18. Jefferson Medical College Hospital, Philadelphia 7, Pennsylvania.

SOUTH CAROLINA ORTHOPAEDIC ASSOCIATION

The Annual Meeting of the South Carolina Orthopaedic Association was held in Hilton Head Island, South Carolina, September 26 and 27, 1959.

A brief résumé of the papers follows:

Opening Remarks. Dr. William A. Boyd[1].

Chronological Pathology of the Hip. Dr. Austin T Moore [2] showed a sound movie, in color, of the first operation of its kind to replace the upper end of the femur with a Vitallium prosthesis. The operation, done in 1940, consisted in removing the upper third of a femur containing a giant-cell tumor and replacing it with a special Vitallium prosthesis. Following this was another short film illustrating the currently used self-locking Vitallium hip prosthesis and the techniques of several different operations. A number of patients were shown before operation and some were shown from two to seven years after operation. The sound track recorded a brief statistical report of approximately 250 cases, the results of which have been considered satisfactory.

Congenital Dislocation of the Hip. Dr. George R. Miller [3] (by invitation).

1. 1524 Pickens Street, Columbia 49, South Carolina.
2. 1528 Gervais Street, Columbia, South Carolina.
3. North Carolina Orthopaedic Hospital, Gastonia, North Carolina.

Transient Synovitis of the Hip. Dr. Edwin H. Martinat [4] defined this disease as a painful, idiopathic self-limited condition affecting the hip in childhood. The name *idiopathic transient coxitis* is suggested since it is probably the least misleading of all the many terms which have been used for this condition.

Dr. Martinat reviewed the world literature and added sixty-five new cases. He believed that the awareness of this disease was important since it was another condition involving the hip, about which our knowledge is insufficient.

The average age of the patient is approximately five years. The laboratory findings vary quite widely in all the various studies. The roentgenograms show only capsular distention, and about 55 per cent of the patients show roentgenographic changes. The treatment is rest. The condition, of course, must be differentiated from various other conditions, particularly coxa plana and rheumatoid arthritis. No definite conclusions as to the etiology or pathology of this condition could be drawn.

The Treatment of Coxa Plana. Dr. John E. Keith [5] presented a study of eighteen patients followed at the Spartanburg Orthopaedic Clinic and seventeen patients at the University of North Carolina.

Satisfactory results were found in 67 per cent of the Spartanburg group and in 76 per cent of the University of North Carolina group. The satisfactory group had been treated with traction in the early stages and an ischial weight-bearing brace in the regenerative stage. Poor results were found in those patients who were untreated or treated late. The following treatment was outlined: (1) traction to relieve all muscle spasm, maintaining a good range of hip motion throughout the disease; (2) continuous non-weight-bearing, either by bedrest or a properly fitted ischial weight-bearing brace and an elevated shoe, until regeneration is well advanced along the weight-bearing stress area of the hip; and (3) bedrest with traction in the early-age group and the group with bilateral involvement.

Epiphyseolisthesis of the Hip. Dr. Frank H. Stelling [6].

Pathology of the Adult Hip. Treatment by Arthrodesis. Dr. John A. Siegling [7] reported on twenty cases of arthrodesis of the hip. All operations had the following in common: (1) the long Smith-Petersen nail transfixing the hip joint; (2) the massive bone graft with or without a plaster cast; and (3) no attempt to dislocate the hip or denude the head of the femur or the acetabulum of cartilage.

The diagnoses before fusion were: malum coxae senilis in eight patients; old pyogenic arthritis in five; old slipped epiphysis in three; dislocation with avascular necrosis in two; and fracture with avascular necrosis of the head in one.

Seventeen patients were followed from two to fifteen years. One died of cerebral thrombosis four months after surgery. Another was killed riding a motorcycle seven months after fusion. The remaining fifteen had solid fusion. Three patients had sustained subtrochanteric fractures of the femur since arthrodeses of the hip.

The Mechanism of Cervical-Spine Injury. Dr. H. Francis Forsyth [8] said that the incidence of injury to the cervical spine has increased in recent years. The most important aid to good treatment of these serious injuries is an accurate concept of the mechanism of injury in each case. One must take the time to get a detailed history of the accident. He must pay close attention to the position of abrasions and lacerations about the head. He must have adequate roentgenograms and know how to interpret them. I have analyzed 159 cases of serious injury to the cervical spine and classified them according to the mechanism of injury. Each characteristic type of injury is caused by a certain type of force acting in a certain direction, producing a result that is highly predictable.

The primary forces involved are flexion and extension, depending on whether the blow is delivered to the back of the head or the front. These forces may be modified by having a downward component referred to as "compression", or an upward direction in which case we say it is "without compression". If a direct force is applied in the mid-line the resulting injury will be symmetrical or "bilateral", but if the force is tangential or is applied to one side of the head, the resulting injury will be of a "rotatory" or "unilateral" nature. Occasionally, a pure compression force is applied by a fall directly on the top of the head, with no element of flexion or extension. This results in a "bursting fracture" which usually involves the atlas. The most significant fact that shows up in my analysis is that "extension forces" are now known to cause at least as many of the demonstrable injuries of the cervical spine as the "flexion forces". This is contrary to any previous thinking on the matter.

4. 9–11 Medical Court, Greenville, South Carolina.
5. 201 Professional Building, Spartanburg, South Carolina.
6. 9 Medical Court, Greenville, South Carolina.
7. 70 Ashley Avenue, Charleston 37, South Carolina.
8. Bowman Gray School of Medicine, Winston-Salem 7, North Carolina

The simple and practical classification which covers almost every conceivable type of injury to the cervical spine is as follows:

I. Flexion: (Unilateral)
(Bilateral)
A. Without compression
(Dislocation)
B. With compression
(Fracture or fracture-dislocation)
C. Hyperflexion
II. Extension: (Unilateral)
(Bilateral)
A. Without compression
(Posterior dislocation)
B. With compression
(Fracture or fracture-dislocation)
III. Combined flexion and extension
IV. Compression (bursting fracture)
V. Direct trauma

Dr. Forsyth's discussion was followed by a series of slides with diagrams depicting the typical mechanism of each major type of injury and an illustrative roentgenogram of that type.

SOUTHERN MEDICAL ASSOCIATION

SECTION ON ORTHOPAEDIC AND TRAUMATIC SURGERY

As Prepared by H. Robert Brashear, M.D., *Section Secretary*

The 1959 meeting of the Section on Orthopaedic and Traumatic Surgery of the Southern Medical Association was held in Atlanta, Georgia, November 17 through 19, 1959, under the Chairmanship of A. H. Weiland, M.D., Coral Gables, Florida.

Officers of the Orthopaedic Section of the Southern Medical Association elected for 1959-1960 included Daniel C. Riordan, M.D., *Chairman;* Elias Margo, M.D., *Vice-Chairman;* and H. Robert Brashear, M.D., *Secretary.*

Evaluation of Intramedullary Fixation of Tibia with Lottes Nail. Dr. Loui G. Bayne[1], Dr. Jack Wickstrom[2], and Dr. Harry Morris[3] presented a series of fractures of the middle third of the shaft of the tibia collected at the Charity Hospital of Louisiana.

There were forty patients with forty-one fractures of the tibia. The fractures selected for this study included open as well as closed fractures. Fractures of the middle third which were satisfactorily reduced and held with plaster immobilization, as well as oblique fractures which were suitable for screw fixation, were omitted from this series. The series included four cases of nonunion of the tibia in which bone grafts were used in combination with intramedullary fixation.

The main complications were two infections, which occurred in closed fractures and were considered a complication of surgery.

It was found in this small series that intramedullary fixation was relatively easy and a safe way to treat fractures of the middle third of the tibia. Patients with transverse and oblique fractures bore weight sooner by this method. In severely comminuted fractures, intramedullary fixation was a great aid in maintaining the alignment of the fracture and early mobilization; however, the authors stated that when closed reduction and plaster immobilization yield satisfactory position of the fracture, this is their choice of treatment.

The Management of Skeletal Fractures in the Patient with a Head Injury. Dr. J. B. Wray[4] and Dr. C. H. Davis[5] presented a series of seventy-five patients with fractures and with significant head injuries. A total of 146 fractures was encountered. Of these, almost one-half were seen in the lower extremity and one-third in the trunk and spinal axis. Eight of the fractures were open. Head injuries ranged from cerebral laceration to contusion with or without hematoma formation. Cases of cerebral concussion only were not included in this series.

Three considerations were found of particular importance in the successful management of

1. 340 Boulevard, N. E., Atlanta 12, Georgia.
2. 1430 Tulane Avenue, New Orleans 12, Louisiana.
3. 3503 Prytania Street, New Orleans 15, Louisiana.
4. Bowman Gray School of Medicine, Winston-Salem 7, North Carolina.
5. Bowman Gray School of Medicine, Winston-Salem 7, North Carolina.

these seriously injured patients. First, a thorough musculoskeletal examination is imperative to avoid overlooking less obvious injuries. Second, treatment to the musculoskeletal system should be minimized in the early hours after injury when the added insult of anesthesia and extensive treatment may prove fatal. Third, definitive orthopaedic treatment of a major nature should be deferred until vital body functions have stabilized and until significant organ damage has been dealt with.

Giant-Cell Tumor of Bone. Dr. John S. Forr[6] and Dr. J. L. Goldner[7] discussed several cases of giant-cell tumor, including two unusual lesions of the pelvis. The authors said that treatment should be directed toward total excision, when possible. Excision and extensive curettage, followed by grafting, may be necessary to preserve function when the lesion is directly adjacent to articular cartilage. This method, however, will probably result in a higher recurrence rate than if total excision is possible. Radiation therapy is potentially hazardous and should be reserved for lesions which are inaccessible surgically. Sixteen patients had been treated at Duke Medical Center; the follow-up was adequate for them to be included in the review. More than one operation was required in eight patients and three patients in this group died as result of the tumor. Amputation was necessary in five of sixteen patients. The review emphasized the necessity of a guarded prognosis, the need for close, careful, continuous follow-up, and the inherent aggressiveness of giant-cell tumor of bone.

Lest We Forget. Dr. Arthur Weiland[8], *Section Chairman*, gave a paper dealing with the events leading up to the invasion of France on D Day, June 6, 1944. It stressed particularly the so-called vertical force type of injury encountered in the underwater explosion of the acoustic mines under the fantail ships carrying personnel. Lantern slides of exceedingly severe injuries of the lower extremities were shown, including a series of twenty cases of complete dislocation of the knee. Total number of casualties observed—847.

Dual Bone Graft for Congenital Pseudarthrosis of the Tibia—Variations and Technique. Dr. George D. Purvis[9] and Dr. E. J. Holder[10] described a two-stage procedure consisting in excision of the pseudarthrosis and any necessary tendon-lengthening and fixation of the fibula with an intramedullary pin; followed by the second stage, consisting of an autogenous cortical graft, taken from the opposite tibia, used to bridge the tibial defect. This had been used in seven cases, six of which had been followed for over eighteen months and had all united. The follow-up period was from eighteen to ninety-nine months, with an average period of follow-up of four years and seven months. This technique appears to offer a better prognosis for union. It is a method which can be used early enough to decrease the tendency for shortening and calcaneus deformity.

Surgical Treatment of the Intermediate Club-Foot. Dr. William M. Roberts[11] and Dr. R. W. Gaul[12] discussed a series of twelve patients with congenital club feet who were first seen at the North Carolina Orthopaedic Hospital during the years 1938 to 1947 and in whom recurrences developed when they were from four to eight years old, which required surgery for correction. The operations utilized and the results were reported on ten of the twelve patients, two of the patients being omitted because of inadequate follow-up and surgery performed elsewhere. The complete series represents 12 per cent of all the patients with congenital club feet admitted to the North Carolina Orthopaedic Hospital in this ten-year period. Follow-up study of the ten cases reported indicated that bone remodeling is necessary to obtain a satisfactory result. The technique of the club-foot plastic operation was presented. The operation is similar to triple arthrodesis but the articular surfaces are preserved.

It is known that the treatment of a congenital club-foot should be conservative but it is felt that surgical remodeling is necessary in a small percentage of recurrent club feet and that a satisfactory foot can be salvaged under these conditions.

A New Treatment of Radio-Ulnar Synostosis. Dr. Louis P. Brady[13] and Dr. Eugene L. Jewett[14] discussed traumatic synostosis occurring at the proximal end of the radius and ulna. A technique was described employing an ordinary bone screw to displace the radius away from the ulna after osteotomy near the proximal end of the radius. By this procedure the full physiological width of the interosseous membrane was maintained and the prime factor in the causation of synostosis—the proximity of bone to bone—was removed. No fascia lata was used and gelfoam was placed in the depths of the wound covering the osteotomy in the radius. A plaster cast, with the elbow at right angles with the palm and wrist incorporated, was left in place three to six weeks,

6. Duke University School of Medicine, Durham, North Carolina.
7. Duke University School of Medicine, Durham, North Carolina.
8. 263 Aragon Avenue, Coral Gables, Florida.
9. 514-D East Woodrow Wilson Avenue, Jackson 6, Mississippi.
10. Mississippi Baptist Hospital, Jackson 2, Mississippi.
11. North Carolina Orthopaedic Hospital, Gastonia, North Carolina.
12. Hawthorne Medical Center, Hawthorne Lane, Charlotte 4, North Carolina.
13. 502 East Colonial Drive, Orlando, Florida.
14. 502 East Colonial Drive, Orlando, Florida.

following which the screw was removed and active physical therapy begun. Two cases of synostosis were presented with the results of treatment; in another case the screw was used prophylactically. It was felt that this method offers a simple atraumatic technique of removing one of the major factors leading to synostosis and should be useful both from a prophylactic and a treatment standpoint.

Indications and Techniques for Early Stabilization of the Neck in Some Fracture Dislocations of the Cervical Spine. Dr. Robert A. Robinson [15] and Dr. Wayne O. Southwick [16] discussed unstable cervical-spine fractures. The protection afforded by the intact cervical spine to the spinal cord, the cervical-nerve roots and their ganglia, and the vertebral arteries is not only lost but the safety of these soft-tissue structures is actively threatened when the bone segments of the cervical spine become unstable. When reduction of a fracture-dislocation has not been achieved by skull traction, and an open reduction and laminectomy is to be undertaken; or when, despite reduction of the osseous deformity, a laminectomy is indicated to relieve pressure on the soft tissues enclosed in a cervical spine; then it is possible to do three things at once: an open reduction, a laminectomy, and internal stabilization of the cervical spine. Internal stabilization after fracture-dislocation is, it appears, as important as reduction of the osseous deformity and relief of bone pressure on the soft tissues in the cervical spine for the final achievement of the best clinical result.

Two techniques for accomplishing internal stabilization of the neck simultaneously with laminectomy and open reduction were described. The first technique is indicated in flexion fracture-dislocations in which stainless-steel wire reins running from the inferior facets of the dislocated vertebra and the vertebra above to the posterior spinous process of the seventh cervical or the first thoracic vertebra maintain the reduction until anterior disc removal and interbody fusion can be performed about ten to fifteen days later.

The second technique can be used for either flexion or extension fracture-dislocations of the cervical spine. Tibial or iliac bone grafts are fixed to the facet joints on either side of the laminectomy site. These grafts extend one or two levels above and two levels below the level of dislocation and the struts are held to the facet joints by loops of stainless-steel wire which pass through the inferior facets and around the grafts at each of these vertebral levels.

The case histories and roentgenograms of five patients were presented to show the clinical application of these two operative techniques.

Spondylolisthesis and Spondylolysis. Dr. Elias Margo [17] presented a clinicostatistical report on 589 cases of spondylolisthesis and spondylolysis seen in the McBride Clinic and Bone and Joint Hospital over a period of fourteen years (1945 through 1958). This included a review of 153 surgical cases and 436 non-surgical cases. A classification of the type of case—whether private, liability, or industrial, as well as according to sex—was presented. It was concluded that conservative treatment is essential at the beginning. Operative treatment is indicated: (1) in the youth when conservative measures fail; (2) in patients with recurrences in spite of conservative treatment, especially among laborers; (3) for severe cases, when fusion is the logical treatment; (4) in increased lordosis, when lumbar fasciotomy as an adjunct aids particularly; and (5) at disc removal, when herniation is present as was the case in thirty-nine patients.

Technical Faults and Operative Therapy for Fractures of the Hip. Dr. Carl Badgley [18] (Guest Speaker).

Therapy of Clinical Fat Embolism with Heparin. Dr. Cully A. Cobb, Jr. [19], Dr. J. William Hillman [20], Dr. C. B. LeQuire [21], and Dr. V. S. LeQuire [22] presented a series of twenty cases of fat embolism complicating long-bone fractures. There were sixty-four fractures. In 70 per cent of the patients, fractures were multiple; in 95 per cent the femur or the tibia or both were involved. A clinical syndrome of delirium, stupor, pyrexia, and decerebrate rigidity in the presence of marked tachycardia was observed in each patient. The authors believe that the diagnosis of clinical fat embolism is best reserved for patients showing this syndrome in some degree.

Heparin in small doses was used in the patients showing signs of progressive cerebral involvement, without complication and with favorable effect. Eighty-five per cent of the patients are now fully recovered.

Experimental studies indicated that a period of four to five days is required for mobilization of embolic fat. In patients with multiple fractures staging of manipulations by such periods is recommended. A more rapid mobilization of experimental emboli was observed in animals treated with heparin.

15. Johns Hopkins Hospital, Baltimore 5, Maryland.
16. 789 Howard Avenue, New Haven 4, Connecticut.
17. 605 N. W. Tenth Street, Oklahoma City 3, Oklahoma.
18. 1313 East Ann Street, Ann Arbor, Michigan.
19. Vanderbilt University School of Medicine, Nashville, Tennessee.
20. Vanderbilt University School of Medicine, Nashville, Tennessee.
21. Vanderbilt University School of Medicine, Nashville, Tennessee.
22. Vanderbilt University School of Medicine, Nashville, Tennessee.

Polymer Osteosynthesis. Dr. G. G. Brown [22], Dr. W. V. Trekell [24], and Dr. Irving Redler [25] discussed osteosynthesis by means of a polyurethane polymer. This was attempted fifty times on forty-one patients, eight of whom were children. The group included six fresh and pathological fractures and ten non-unions and arthrodeses. The basic operative technique consisted in preparation of a cavity at the site of desired bone fixation, filling it with the liquid polymer, and maintaining fixation until the material hardened. Weight-bearing or functional use was permitted as soon as the wound was healed. Of the fifty operations, healing by callus formation occurred on the side opposite the polymer in eleven, failure occurred in seventeen because the polymer column broke or pulled away from the bone cavity, and fixation was maintained externally in twenty-two, but without any roentgenographic evidence of healing.

Wound infection and draining sinuses developed in fourteen patients. It was necessary to remove the polymer in ten patients in order to stop the persistent drainage. This complication is probably caused by the foreign-body reaction to the polymer.

Microscopic study of removed specimens of the polymer showed fibroblastic invasion of the superficial portions. There has been no evidence of osteoblastic replacement of the polymer to date.

Further study of the patients on whom operation was done will be continued. Future use will be restricted to pathological fractures and situations not amenable to conventional methods.

Arthroplasties Using Compressed Ivalon Sponge. Dr. M. C. Cobey [26] and Dr. A. R. Crain [27] described compressed ivalon sponge which is made by autoclaving ivalon sponge as supplied by the surgical house and compressing it to the desired thickness between two sheets of aluminum. The ivalon sponge can be compressed to the desired thickness of one-eighth of an inch recommended for arthroplasties. This has been used in most of the joints in the body. Because of its thinness it produces less synovial response but at the same time retains its good qualities of being held in place by the invasion of fibrous tissue from the edges of the joint surface. It is also kept moist by the joint fluid. Because of its rubbery and more compressed nature, the motions of the joint and the weight-bearing pressures do not cause the sponge to shred and wear out, thus obviating the necessity of replacing the sponge after several years of stress and strain, such as severe activity.

Fractures of the Forearm. Dr. W. J. Jinkins, Jr. [28], presented a study of 111 consecutive cases of fractures of the forearm in adults treated at the University of Texas Medical Branch. There was a 7.2 per cent rate of non-union in 111 fractures. Three were reopened and bone grafts were applied; union occurred. The rest were lost to follow-up, with a final non-union rate of 4.5 per cent. Synostosis occurred in 2.9 per cent, postoperative infection in 1.8 per cent, and neurological complications in 0.9 per cent.

The ulna is relatively fixed at the elbow by the olecranon in the trochlear groove as regards pronation and supination, and greater torsional strain is expected at the fracture site with a higher non-union rate in the ulna. As a result of this shearing force, the ulna is not suited for intramedullary-rod fixation. Those patients in whom the lateral curvature of the radius had been lost had a diminished range of pronation and supination—thus, this lateral convexity should be preserved. Contact compression and continuity are necessary for rapid bone union. Iliac grafts in the form of long strips were used in fresh segmental fractures, severely comminuted fractures, and all non-unions.

A four-inch narrow contact splint is always used in the ulna and a three or four-inch contact splint or intramedullary rod is used in the radius. The screws in the contact splint are put in tightly and then loosened to a quarter to a half a turn to allow compression. Anterior and posterior plaster splints are used for immobilization during the first ten days, then a circular cast is applied from the metacarpophalangeal joints to the upper one-quarter of the humerus. The web between the thumb and index finger is not included in the cast.

Tendon-Lengthening for Volkmann's Ischemic Claw-Hand. Dr. J. Kulowski [29] discussed the ischemic contracture of Volkmann which affects chiefly the extrinsic (long) muscles and tendons of the hand and wrist. The clinician must distinguish between a paralytic and an ischemic condition and also recognize any participation of the intrinsic muscles in order to choose the best operative treatment (after failure of preliminary non-operative methods). Surgical methods are essentially muscle-slackening ones, with the exception of excision of necrotic muscle bellies joint fusion (wrist), and booster tendon transplantations. The author recommended tendon-lengthening (Z-method) of all of the affected flexors, which has given satisfactory results not only in Volkmann's but similar types of deformity. He illustrated this method by three cases, observed for three, ten, and twenty years after operation, respectively. These cases also demonstrated the need for treating other associated lesions, which contributed to the ultimate good results obtained.

23. 1539 Delachaise Street, New Orleans 15, Louisiana.
24. Louisiana State University, New Orleans, Louisiana.
25. 1539 Delachaise Street, New Orleans 15, Louisiana.
26. 1726 Eye Street, N. W., Washington 6, D. C.
27. 1726 Eye Street, N. W., Washington 6, D. C.
28. Orthopaedic Division, Medical Branch, University of Texas, Galveston, Texas.
29. 422 Felix Street, St. Joseph, Missouri.

Evaluation of the Colles' Fracture. Dr. Wallace Miller [30] discussed Colles' fracture and illustrated a method of management.

Tendon Transplants at the Wrist for Nerve Injuries. Dr. William Minor Deyerle [31] and Dr. Fred Tucker [32] presented a long-term follow-up of from two to nine years of tendon transplants at the wrist. There were a total of seventeen cases; ten of them involved radial-nerve injury and were presented in detail.

It is advised that in open wounds, where it is possible, that the nerve should be carefully evaluated and a note made on the record of the condition both from a functional standpoint and from a direct view through the open wound when possible. In trauma in which there is no open wound with total immediate wrist-drop, after careful clinical and electromyographical studies are made, if the status of the nerve remains in doubt exploratory operation should be done to determine whether or not the nerve is severed. This is necessary because we have seen contused nerves at surgery require as long as a year and a half to return, and if one knows that the nerve is merely contused he can wait with a little more assurance and confidence. An avulsed or severed nerve returns in such a small percentage of cases that the authors advise routine tendon transplants to maintain function and pliability with the understanding that in an occasional case this may be reversed. Four of our ten cases had nerve repair and never showed any regeneration, justifying the early tendon transplant. The results of the ten cases of radial-nerve injury with multiple tendon transplants through multiple incisions were excellent in four, good in four, fair in two, and poor in none. The result of the other seven transplants, although there were various types of injury, was improved function to the extent that both the surgeon and the patient felt that the operative procedure was worthwhile. Since there were so few of these cases, they were not reported in detail.

The Athlete and His Knees. Dr. Jack Hughston [33] stated that many an injured knee can be saved by teamwork among the coach, trainer, team physician, and orthopaedic consultant. Diagnosis is aided greatly by knowing the mechanism of injury. Ligamentous injuries are more easily diagnosed within the hour. Later evaluation may be falsified by protective muscle spasm.

Treatment of fresh ligamentous injuries is immediate. Major tears need surgical repair and immobilization. In meniscus injuries, recurring difficulty demands non-athletic activity until operative correction at an elective date. Adequate operative exposure of the knee with removal of all damaged tissue was stressed.

Twenty-five of 100 patients operated on exhibited chondromalacia of the patella. Forty-two demonstrated a torn anterior cruciate ligament, of which only one-third of those with a torn anterior cruciate ligament had a positive Drawer sign. One-half of those with a torn anterior cruciate ligament returned to full athletic activity without symptoms of pain, swelling, or giving way.

Treatment of Leg-Length Discrepancy. Dr. Warren G. Stamp [34] and Dr. W. Edward Lansche [35] discussed one hundred and forty patients with leg-length discrepancy who were surgically treated between 1937 and 1957 at the St. Louis Unit of the Shriners' Hospital for Crippled Children. There were fifty-nine girls and sixty-nine boys (128 patients) who were followed until their growth was complete. The average age at the time of follow-up was nineteen years for girls and twenty for boys.

Surgical treatment was divided into four groups: 102 Phemister-type epiphyseal arrests; twelve Blount staplings; twelve leg-lengthenings; and twenty leg-shortenings. Sixty per cent of the Phemister-type epiphyseal arrests were regarded as being successful, while 80 per cent of the staplings were successful. Both the leg-lengthening and the leg-shortening procedures were successful in approximately 75 per cent of the cases. Four per cent of the patients in whom epiphyseal arrest was done required osteotomies to correct growth disturbances secondary to the epiphyseodesis. In all of the patients, the anticipated height was determined clinically from the family history, and the amount of epiphyseal growth anticipated was calculated on the assumption that 40 per cent of the growth would occur in the distal portion of the femur and 30 per cent in the proximal portion of the tibia.

As a result of this study, a chart has been added to the permanent record of each child so that the pertinent data may be recorded, and, in addition, several methods of growth determination are being used (W. T. Green and J. Warren White). The permanent type of epiphyseal arrest is a satisfactory way to control leg-length discrepancy if the surgery is done carefully and early enough. Leg-shortening should be considered in those patients who have considerable shortening near the end of the growth period. Leg-lengthening is rarely indicated.

Management of the Neuropathic Knee. Dr. J. Cordrey [36] presented a movie covering the history, pathology, and clinical findings in neuropathic joints. A modification of the Charnley arthrodesis was also shown.

30. University of Miami, School of Medicine, Miami 36, Florida.
31. 2222 Monument Avenue, Richmond 20, Virginia.
32. Clearwater, Florida.
33. 1316 13th Avenue, Columbus, Georgia.
34. 600 S. Kingshighway, St. Louis 10, Missouri.
35. 600 S. Kingshighway, St. Louis 10, Missouri.
36. 4801 Woodmere Avenue, Tampa 9, Florida.

LETTER TO THE EDITOR

To the Editor:

The authors of the article "The Talar-Tilt Angle and the Fibular Collateral Ligament", published in *The Journal of Bone and Joint Surgery*, Vol. 42-A: 311–326, March 1960, would like to call your attention to the following errors on their part which they overlooked in the original paper and in the proofs but, perversely, noted in the published article:

1. Page 311, paragraph 3, line 5: reference number 20 after the word *ankle* should have been reference number 8.

2. Page 311, the paragraph under the heading *Definition:* the figure referred to should have been Fig. 2, not Fig. 3.

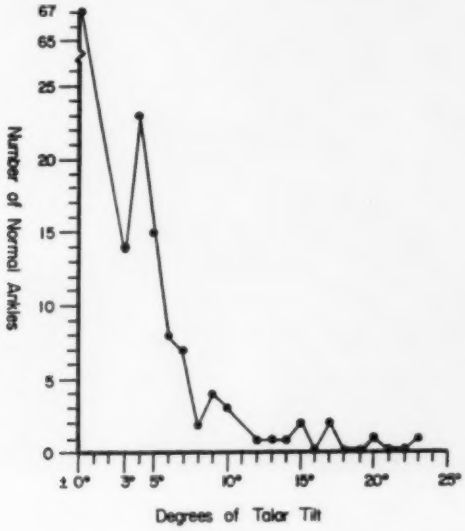
3. Page 321, Chart I: a dot should be placed on the curve of the Chart as it crosses the intersection of the following coordinates:

a. 15 normal ankles and 5 degrees of tilt;

b. 3 normal ankles and 10 degrees of tilt;

c. 1 normal ankle and 13 degrees of tilt.

The curve should indicate 14 cases with 3 degrees of tilt instead of 12 cases. The accompanying chart incorporates these corrections.



4. Page 322, Chart II: a dot should be placed on the curve of the chart as it crosses the intersection of the coordinates for 2 sprained ankles and 13 degrees of tilt.

With the charts corrected as described each of the cases in these categories may be easily located.

Gustav Rubin, M.D.

WILLIAM BENJAMIN MAC CRACKEN
1907 — 1960

On February 29, 1960, Dr. William Benjamin MacCracken died.

Born on September 2, 1907, in Mt. Vernon, New York, where Dr. MacCracken also completed his primary education, he was graduated from New York University in 1928 and from the Columbia College of Physicians and Surgeons in 1932. Following his internship at the Bellevue Hospital, he completed his residency at the New York Orthopaedic Dispensary and Hospital and was subsequently appointed as Annie C. Kane Fellow for three years at the same institution. Upon completion of his research fellowship, he established his practice in orthopaedics in Oakland, California.

The call of the sea was strong, and the war clouds were gathering, with the result that in September 1941, Dr. MacCracken enlisted in the United States Navy as a Lieutenant. He was retired from the Naval Service with the rank of Commander and subsequently became associated in the practice of orthopaedics with Dr. Francis Scott of Huntington, West Virginia, on February 1, 1946. Dr. MacCracken was in active practice until the time of his death. He was a Member and Past-President of the Russell A. Hibbs Society, a Member of The American Academy of Orthopaedic Surgeons and The Tri-State Orthopedic Society, as well as several other regional and local orthopaedic organizations.

He was Chief Orthopaedic Surgeon for the Chesapeake and Ohio Railroad. His contributions to the orthopaedic literature were not many, but the few he did write were succinct and of excellent educational value.

Bill, or "Willie", as he was more affectionately called by his very close friends, was a quiet, modest, and, at times, almost painfully taciturn individual, but behind this façade there was an affectionate heart that asked for nothing and gave everything. The year 1942 and the dates August 17 and 18 are not too far removed from the memory of many of us, nor the submarine known as the "Nautilus", nor the Second Raider Marine Battalion also known as "Carlson's

Raiders". Bill was one of them. As if this assignment in itself were insufficient to prove his courage and his selfless devotion to his country and to his fellow-men, Bill, on those two memorable days, so distinguished himself that he was decorated with the Navy Cross by Admiral Nimitz. His citation read as follows:

"For extraordinary heroism and distinguished service above and beyond the call of duty as Senior Medical Officer of the landing force of the Marine Raider Battalion against Japanese held Makin Island August 17–18, 1942.

"Following the first enemy aerial attack, Lieutenant MacCracken personally carried many of the wounded to positions of greater safety, and by his complete disregard for his own life and this gallant action, he was enabled to administer early and effective aid. When his boat overturned, endangering the life of a seriously wounded private during the first attempt at evacuation, he risked his own life to save that of another by holding the helpless man's head above water and swimming ashore with him. After returning to his ship,* Lieutenant MacCracken performed six major operations under most difficult circumstances and, as a result of his skillful and tireless efforts, he succeeded in bringing all of his cases back to the base line in excellent condition. His skill, courage and fine sense of moral obligation to those entrusted to his professional care were in keeping with the highest traditions of the United States Naval Service." Signed, Chester W. Nimitz, Admiral.

He was subsequently decorated a second time by Major-General A. A. Vandergrift of the United States Marine Corps for outstanding service during the invasion of Aola Bay on November 5, 1942. He received other unit citations as well during the various operations of the Second Raider Marine Battalion throughout the South Pacific Area.

Such a man was Bill.

He is survived by his wife, Myrtle May MacCracken, his mother, Mrs. John A. MacCracken, and his daughter, Mrs. James L. Murphy. To them we extend our deepest sympathy for their tragic loss. To those of us who admired him and to whom he was affectionately known as "Willie", he will always be remembered as a man's man, a lover of the out-of-doors and of the camaraderie it brought, and as an intense, sincere, painfully honest orthopaedic surgeon who compromised for nothing but perfection.

N.J.G.

* The ship was the submarine, "Nautilus".

News Notes

WESTERN ORTHOPEDIC ASSOCIATION

On the cover of the June issue of The Journal there appears for the first time the name of the **Western Orthopedic Association.** Pursuant to a recently signed agreement between The Journal and this large regional society, with its total membership of over seven hundred orthopaedic surgeons, The Journal is now the official organ of the "Western". It is a pleasure to welcome this active and productive society to The Journal family.

The Proceedings of the **Japanese Orthopaedic Association,** published in the April issue of The Journal, were edited by Tadao Ishizuka, M.D., Bun i'chiro Kawamurg, and Hiroshi Ikenchi, M.D., with the assistance of Dr. David M. Bosworth. To them The Journal wishes to express its indebtedness and thanks.

The **St. Louis Orthopedic Society** held its Biannual Meeting at the University Club in St. Louis, Missouri, on April 13, 1960. Guest Speaker for the evening was Dr. Harold Boyd of the Campbell Clinic of Memphis, Tennessee, who spoke on the use of bone grafts in long bones. The new officers elected for the coming year are: Dr. Oscar Hampton, President; Dr. Ralph Earp, Vice-President; and Dr. Richard E. Lord, Secretary-Treasurer.

The **Alabama Orthopaedic Society** held its Ninth Annual Meeting on April 20, 1960, at the Rotary Foundation Diagnostic and Rehabilitation Center, Mobile, Alabama. Twenty-three members and guests were present. Dr. William F. Enneking, Gainesville, Florida, presented a paper on osteogenic sarcoma in which he discussed its treatment, especially in regard to radical local excision. Data were also presented concerning the antigen-antibody properties of these tumors. Dr. Robert T. King, Mobile, Alabama, presented a paper on the use of a Vitallium sphere in the treatment of non-unions and degenerative conditions of the carpal scaphoid. Dr. William J. Hutchison, Tallahassee, Florida, discussed the value of discography as opposed to myelography in the diagnosis of intervertebral-disc protrusions and pointed out the value of injecting the nucleus pulposus with hydrocortisone for the relief of symptoms arising from these protrusions. Dr. D. T. Imrie, Vicksburg, Mississippi, gave his views on the value of epiphyseal stimulation in leg-length inequalities. The Scientific Section was concluded with a discussion by Dr. Kenneth M. Hannon, Mobile, Alabama, of primary prosthetic replacement in acute femoral-neck fractures in the aged. Dr. John E. Hanby was elected President; Dr. E. C. Brock, Secretary-Treasurer; Dr. Paul D. Everest, President-Elect; and Dr. Mervel V. Parker, Secretary-Treasurer-Elect. The 1961 meeting of the Alabama Orthopaedic Society will be held in Tuscaloosa, Alabama.

The Thirty-fifth Anniversary Congress of the **Pan American Medical Association** was held in Mexico City, May 2 through 11, 1960.

Dr. Joseph J. Eller, Director General of the Association, announced a program of more than 900 scientific papers, ranging in subject matter from space medicine to clinical hypnosis.

President Adolfo Lopez Mateos of Mexico opened the Congress on May 2 in ceremonies at the National Auditorium. Dr. Alfonso Asenjo, Santiago, Chile, is President of the Association, which has as its credo "The practice of medicine has no national, racial or religious boundaries".

Honorary President of the Congress is Dr. Jose Alvarez Amezquita, Minister of Health of Mexico. Dr. Clemente Robles, Mexico City, was named President of the ten-day scientific Congress.

There has recently been formed the **Atlantic Provinces Orthopaedic Society** which includes the Canadian Provinces of New Brunswick, Nova Scotia, Prince Edward Island, and Newfoundland. This Society was formed on January 16, 1960, at a meeting held in Moncton, New Brunswick, despite a very severe snowstorm which kept away many members who would have attended. There were representatives from three of the four Provinces.

The officers chosen are: Dr. E. W. Ewart, Moncton, New Brunswick, President; Dr. T. B. Acker, Halifax, Nova Scotia, Vice-President; and Dr. K. Seaman, Saint John, New Brunswick, Secretary-Treasurer.

The object of this Association is stated to be the advancement of the art and science of ortho-

paedic surgery in all its phases, with particular reference to the problems of practice and training in the Atlantic Provinces.

The Part II examination for Certification by the **American Board of Orthopaedic Surgery** will be held January 4 through 6, 1961, at the Americana Hotel, Bal Harbour, Miami Beach, Florida. Sam W. Banks, *Secretary*, 116 South Michigan Avenue, Chicago 3, Illinois.

The **Exchange Fellowship Committee** of **The American Orthopaedic Association** is now inviting applications from American orthopaedic surgeons who are interested in the tour to Great Britain in the Spring of 1961. The requirements are: The applicant must be under forty years of age as of December 1, 1960; he must have completed Part II of the American Board of Orthopaedic Surgery examinations; and he must be a citizen of the United States. Requests for application blanks should be made to the Chairman of the Exchange Fellowship Committee, Dr. J. Neill Garber, 806 Hume Mansur Building, Indianapolis, Indiana.

KAPPA DELTA AWARD—1961

The Committee on Scientific Investigation of The American Academy of Orthopaedic Surgeons announces that all manuscripts which are to be submitted for the Kappa Delta Award, which is presented annually for the outstanding scientific contribution in the field of orthopaedic surgery and which carries an honorarium of $1,000.00, should be received by June 15, 1960. Manuscripts should be forwarded to the Chairman, Robert D. Ray, M.D., University of Illinois College of Medicine, 840 South Wood Street, Chicago 12, Illinois.

The winner of the award will be asked to present his paper at the Annual Meeting in January 1961.

The **Eighth Congress** of the **International Society of Orthopaedic Surgery and Traumatology** will be held at the Hotel Astor, New York, N. Y., September 4 through 9, 1960. For information write: Lee Ramsay Straub, M.D., 535 East 70th Street, New York 21, N. Y.

THE FIFTH INTERNATIONAL POLIOMYELITIS CONFERENCE

In Copenhagen, Denmark, July 26, 27, and 28, 1960, the Fifth International Poliomyelitis Conference will be held under the sponsorship of the National Foundation and the Danish Infantile Paralysis Association. The general chairman of this conference is Dr. E. Juel Henningsen, Deputy Minister of Health of Denmark. The Honorary Presidents of the conference are Dr. Jacob Ørskov of Copenhagen and Mr. Basil O'Connor of New York. The meetings will take place at the Falkonercentret, Copenhagen, and the official hotel will be the Hotel 3 Falke.

Because of the great advances in the prevention of poliomyelitis, chief attention during this conference will be given to that subject. The opening section will deal with over-all viruses 1960.

The second section will be a discussion of the present status of the killed or inactivated virus vaccines which will include Efficacy, Duration of Immunity, Standardization of Potency, and Recent Developments. This section will be under the leadership of Professor Stuart Harris, Royal Hospital, Sheffield, England.

The third section will deal with all the aspects of the live attenuated virus vaccine. The first session of this section will be under the leadership of Dr. Andre Lwoff, Pasteur Institute, Paris, and will deal mainly with the Experience in Field, Safety, Efficacy, with participation by representatives from the Pan-American Sanitary Bureau, the Ministry of Health of the U.S.S.R. and the Department of Communicable Diseases of the World Health Organization.

The second session of this section will be presided over by Gunnar Olin, M.D., Director of the National Bacteriological Laboratories, Stockholm, Sweden, and will cover Antibody Responses, Influence of Age, Influence of Dosage, and Regimen, together with the Influence of Interfering Enteroviruses. Finally, the third session of the live virus section will deal with the Effects of Virus in the Community; in this session there will be a special panel of all the investigators concerned with the production of attenuated vaccines from the United States, the U.S.S.R., Czechoslovakia, Holland, Poland, Costa Rica, and the Belgian Congo. Every aspect of the use of attenuated vaccines will be given thorough review.

In concluding the conference, Dr. Frank L. Horsfall, Jr., newly elected President of Sloan-Kettering Institute, New York, will summarize the conference, indicating the problems that still remain in vaccination against poliomyelitis.

Further information concerning the conference may be obtained by writing to the secretariat:

Mr. Stanley E. Henwood, *Executive Secretary*, International Poliomyelitis Congress, 120 Broadway, New York 5, N. Y.

The **Tennessee State Orthopaedic Society** held its Twelfth Annual Meeting at the Baroness Erlanger Hospital in Chattanooga, Tennessee, on March 12, 1960. Forty-four guests and members were present. The following papers were presented: "Interesting Spinal Lesion", by Dr. Bennett W. Caughran; "Complications of Pelvic Fractures", by Dr. Paul C. Thompson; "Spinal Stabilization—Knowles Procedure", by Dr. Richard B. Donaldson; "Eosinophilic Granuloma of the Ribs", by Dr. W. Houston Price; "Internal Derangement of the Metacarpal Phalangeal Joint of the Finger", by Dr. John J. Killeffer; and "Total Scapulectomy and Osteochondritis Dissecans, Femoral Head", by Dr. Robert C. Robertson. Dr. Orren D. Baab, Chicago, Illinois, the Guest Speaker, spoke on "Experiences in the Use of Cultured Calf Bone", and Dr. A. R. Shands, Jr., Wilmington, Delaware, spoke on "Femoral Torsion and Its Importance". Dr. Robert B. Brashear was elected President; Dr. Paul E. Spray was elected Vice-President; and Dr. William F. Gallivan was elected Secretary-Treasurer. The 1961 Meeting will be held in Knoxville, Tennessee.

The Thirty-fifth Semiannual Meeting of the **Tri-State Orthopedic Society** was held at the Homestead Hotel, Hot Springs, Virginia, April 29 and 30, 1960.

The Seventh Annual Meeting of **The Society of Nuclear Medicine** will be held at the Stanley Hotel, Estes Park, Colorado, June 22 to 25, 1960. Over seventy speakers will present new scientific information covering every phase of research, medicine, and surgery as it concerns the use of nuclear phenomena in the diagnosis and treatment of disease. Special emphasis will be given to the diagnosis and treatment of thyroid disease, the therapeutic use of radioisotopes, and the "tools of the trade". The First Annual Address of the Nuclear Pioneers Series will be delivered by Dr. Edward Teller of the University of California. This first Address will be in honor of the late Dr. Ernest O. Lawrence.

Book Reviews

GOUTY ARTHRITIS AND GOUT. AN ANCIENT DISEASE WITH MODERN INTEREST. Thomas E. Weiss, M.D., and Albert Segaloff, M.D. Springfield, Illinois, Charles C. Thomas, 1959. $7.50.

If a need exists for a small textbook on gout, this volume will serve that need adequately. It certainly would be more convenient to have a separate thin volume on one's shelves than to have to turn to the appropriate chapters in cumbersome, large volumes on medicine, pathology, or treatment. However, this is hardly sufficient reason for issuing a new book on such an old subject. One must have other informational reasons. In reading through this book it is not immediately apparent what these reasons are. Perhaps the sections where one should seek new information would be those chapters concerned with physiology and treatment. The section on physiology has the most recent references to the literature, but this section is somewhat too strictly concerned with the metabolism of uric acid in gouty patients and therefore lacks an adequate summary of modern work on purine metabolism as a whole, whatever its relation to gout may be. One would have appreciated a more widely conceived approach to this portion of the subject. Perhaps the single section which makes this book worthwhile is that on treatment. Many of the new, as well as the old, drugs are evaluated in a confident and succinct fashion. Ancillary measures, including surgery, management of complications, and physiotherapy, are well presented. A good word should also be said for the chapter on pathology, which is derived from Dr. Mary Sherman's beautiful collection of specimens. The photomicrographs are superb.

The book is based on the experience with more than 250 patients seen by the authors at the Ochsner Clinic. The authors' experience has certainly been sufficient to allow them to speak with some authority, not only on the classic picture, but also on cases which are so atypical that diagnosis may be difficult. It also gives them a firm basis on which to evaluate critically the methods of therapy now in use. In this respect, therefore, this small volume has definite merit for both reading and reference.

Jonathan Cohen, M.D.

SURGICAL TREATMENT OF SCOLIOSIS. Louis A. Goldstein, M.D. With an Appendix, "Anesthesia in Scoliosis", by D. Vernon Thomas, M.D. Springfield, Illinois, Charles C. Thomas, 1959. $5.50.

Since no subject in the field of orthopaedic surgery presents more controversial facets, it is small wonder that men of wide experience quail at the thought of writing authoritatively about scoliosis. Those who have treated hundreds of patients with scoliosis and have tried numbers of methods of correction tend to develop a receptive attitude toward all variations in sound approaches to this enigma. They also find that no one method of treatment meets the requisites.

The author bases his monograph on experience gained in correcting and fusing thirty-seven idiopathic curves, twenty-three paralytic curves, four congenital curves, and one undesignated curve. He presents a concise description of identification of the primary curve and of the method he employs to select the area of fusion. The method of measurement of the curve, the superimposition of the left bending film upon the correction film to obtain a composite view of all the curves as they will finally appear, and the modification of the Risser turnbuckle cast in which the patient is placed on laterally bent Goldthwaite bars in order to correct first the compensatory curves, all stem from John Cobb. The author has modified this turnbuckle cast to include both thighs and has windowed his casts very widely to expose nearly the entire iliac crest on one side. His description of the details of this cast application and finishing is excellent.

One of the contributions of this volume is the author's description of his technique of fusion, and more especially of his wide exposure of the ilium and his technique of removal of very large amounts of iliac cancellous and cortical bone. There appears to be little doubt that the addition of such large amounts of iliac bone enhances the solidarity of the fusion.

The author does not fuse the facets specifically. He does make a wide exposure to include the transverse processes in the decorticated fusion bed.

The results of surgical treatment are presented. Nearly all of the author's patients were twelve years of age or older. In twenty-five patients with fresh autogenous iliac bone added (during the past six years) there was only one pseudarthrosis (4 per cent). However, he lists in this group one fifteen-year-old boy who lost 20 degrees of correction after the cast was removed. Such loss in this patient in this age group probably does not occur without a graft defect. It matters little whether the graft ultimately becomes solid after loss of correction has occurred—the diagnosis of defective graft must still be made.

In twenty-six patients fused by the addition of banked bone and all other types of grafts there were four pseudarthroses listed (15 per cent). These patients were operated on prior to six years ago.

The author presents a series of well documented case reports including roentgenograms and photographs made both before and after operation. In several of these patients the fusion area has been too short and there has been lengthening of the curve, with a total increase in curve angle.

It is difficult to ascertain whether the author, in his final evaluation, has used the original extent of the curve or the extended curve.

Perhaps the chief criticism of this monograph lies in its failure to include the complexities of scoliosis. To be complete, a volume on scoliosis must include experiences with fusion in the young child, the management of pelvic obliquities in paralytic and congenital scoliosis, the management of the collapsing neck and spine in paralytic scoliosis, and the problem of the patient with deficient pulmonary function.

This monograph is a well written, concise treatise of one method of treatment of relatively uncomplicated scoliosis in the young adolescent. It will be of value to the orthopaedic surgeon who has received little training in the management of scoliosis and to those who have become lost in the maze of conflicting opinions as to treatment of scoliosis. It is not, nor does it pretend to be, a book which covers the whole subject of lateral curvature of the spine.

John H. Moe, M.D.

A Handbook of Clinical Practices Related to Artificial Limbs. Clinical Prosthetics for Physicians and Therapists. Miles H. Anderson, Charles O. Bechtol, M.D., and Raymond E. Sollars. Springfield, Illinois, Charles C. Thomas, 1959. $10.50.

This book is a clinical discussion of amputees and their problems; Book One considers all varieties of upper-extremity prostheses, and Book Two is devoted solely to the above-the-knee amputee.

In the first part is found a detailed analysis of amputee types and basic prosthetic operating principles followed by a standardized system of prescription and check-out of the upper-extremity prosthesis. The principles of harnessing are well presented with adequate diagrams. The unique problems of patients with bilateral upper-extremity amputations are included with guides for their training. Biceps cineplasty is discussed, but some expression of the authors' opinion regarding the value of this procedure would be welcomed.

Book Two introduces the subject of the above-the-knee amputee, with a stimulating discussion of locomotion in normal gait and with the above-the-knee prosthesis. The rationale for use of the ischial-bearing quadrilateral socket is carefully defined, as well as the variations in the other major components of the above-the-knee prosthesis, such as the knee assembly, the type of prosthetic foot, the suspension of the limb, and the all-important alignment of these components. Fabrication and fitting of the quadrilateral socket are explained in sufficient detail to acquaint the physician with the techniques and should serve to make him appreciative of the skill and patience required of the trained prosthetist. Use of the adjustable leg and of the alignment duplication jig are pictured and explained.

This book is a valuable guide and reference for the physician who treats amputees. The generous use of diagrams and photographs clarifies much of this rather specialized technical material.

Richard A. Mayo, M.D.

Instructional Course Lectures. Volume XVI. The American Academy of Orthopaedic Surgeons. Fred C. Reynolds, M.D., Editor. St. Louis, The C.V. Mosby Company, 1959. $16.00.

Each volume of Instructional Course Lectures makes more impressive the contents of the entire series. In 1943, the first year of the lectures, a faculty of fifty discussed eleven major subjects to classes totaling 453. In 1959, a faculty of 181 gave 124 lectures to classes totaling 1,700. Thirty of the 1959 lectures are in this volume; forty-five others have appeared in earlier volumes. In the ten years since the present format was adopted, over 400 lectures have been published. All of these volumes are in print and surely offer one of the richest lodes of orthopaedic lore.

J.A.R.

Pediatric Pathology. Daniel Stowens, M.D. Baltimore, The Williams and Wilkins Company, 1959. $20.00.

Those persons interested in pathology will welcome this book, the first systematized textbook on pathology in children. Even as a first try, it does not come off so well as one would like from the point of view of covering the field, but its novelty and opinionated comments certainly are of interest. It is based primarily on the autopsy material made available to the Armed Forces Institute of Pathology from many sources, most of the material coming from military hospitals. The author, both a pediatrician and a pathologist, was the organizer and registrar of the American Registry of Pediatric Pathology at the Armed Forces Institute of Pathology and now is Pathologist at the Children's Hospital in Los Angeles.

Many good things are included all through this book, which will be useful to the general pathologist and student. In the first chapter there are tables of weight, height, and organ size according to age—very useful data collected from a variety of sources, usually not handy to a pathologist or a pediatrician. In the appendix, over 10,000 cases at the Armed Forces Institute of Pathology from 1954 to 1956 are listed by diagnosis, which provides a good indication of the incidence of the common as well as uncommon causes of death. Between these two extremes is the text proper, which is divided into three parts. The first part concerns infants of the newborn period and includes chapters on prematurity and postmaturity, diseases of the placenta, and erythroblastosis fetalis. The second

part is the account of diseases classified according to etiology. The third part is devoted to the pathology of the different organ systems. Since readers of this Journal are particularly interested in those chapters devoted to pathology of the musculoskeletal system, examples of the defects of this book can be taken from those chapters, even though these same defects are evident to a greater or lesser degree elsewhere. Can any account of septic arthritis in infancy omit the consequences of thrombosis and avascular necrosis on the subsequent development of the hip joint? This one does. Can any account of myositis ossificans disregard the difficulties in differentiating an early lesion from sarcoma? This one says that, histologically, the lesion is unmistakable. The sins of omission weigh heaviest when the book is used for reference. One cannot quarrel with the index, which is excellent, or with the omission of some entities too rare for consideration, but when the details on nearly any specific lesion are looked for in the text, too often they are inadequate and have to be supplemented by the references listed.

Jonathan Cohen, M.D.

BIOLOGICAL PROBLEMS OF GRAFTING. A SYMPOSIUM. Edited by F. Albert and P. B. Medawar. Springfield, Illinois, Charles C. Thomas, 1959. $12.50.

This book comprises the collected papers and discussions of a symposium held under the chairmanship of Professor P. B. Medawar at the University of Liége by The Council for International Organizations of Medical Sciences. The participants, well known workers in the field of tissue transplantation, were from Belgium, the United States, Great Britain, France, Czechoslovakia, Denmark, Sweden, Germany, and Holland.

The only paper specifically related to bone transplantation was by Dr. A. Danis of Brussels University. From his experience with the anterior chamber of the rat's eye as the recipient site, Dr. Danis stated that an autogenous graft of bone marrow, periosteum, or fracture fibrocartilage leads to an "invariably" complete ossicle that seems to have an unlimited life span. He also stated that in homologous transplants, the newly formed bone and marrow unfailingly end in necrobiosis and total absorption of the graft. Dr. Danis explains these findings by stating that it is universally agreed that the destruction of a homologous graft is the result of an immunity reaction set up in the host animal, and that this concept rests on experimental and theoretical bases that are so well established that it would be superfluous to describe them. I cannot agree that such experimental and theoretical bases are so well established, or that it is universally agreed that such a mechanism is the sole cause for the inferiority of bone homografts. Dr. Danis also points to the production and subsequent death of the new bone formed in relation to a homologous graft as evidence that this new bone is derived from the graft, stating that this shows the weakness of the theory of metaplasia by induction.

The remaining papers concern a variety of experimental studies connected with the homograft reaction, and, although of value, are of such a technical nature as to be of little interest except to those workers in this field.

Paul H. Curtiss, Jr., M.D.

ANATOMY: REGIONAL AND APPLIED. R. J. Last, M.B., B.S. (Adel.), F.R.C.S. Second Edition. Boston, Little, Brown and Company, 1959. $15.00.

This new edition of Professor Last's textbook is larger by seventy-four pages than the 1954 first edition. More than half of the added pages are devoted to new illustrations, which now total 372. Other major additions are summaries on the nerves of the limbs to aid in viewing each named nerve as a whole, and new material on microscopic anatomy and on joint movements. The author emphasizes strongly that "the text is intended to be read with the appropriate bones and the prosected part or a museum specimen close at hand for constant reference." Readers of The Journal familiar with his studies of "Specimens from the Hunterian Collection" know how faithfully he follows his own advice.

In discussing the first edition of this book Robert I. Stirling wrote of Professor Last, "The body is to him not an inert cadaver to dissect for topographical information, but a living organism, the form and working of which he explains sometimes on a phylogenetic, sometimes on an embryological and sometimes on a physiological basis, and occasionally from the point of view of a surgeon. Thereby, the reader is provided with numerous pegs on which to hang his knowledge; and finds many parallel approaches, which simplify the understanding of the part and yet emphasise the cohesion of the whole." *J.A.R.*

THE DEGENERATIVE BACK AND ITS DIFFERENTIAL DIAGNOSIS. P.R.M.J. Hanraets, M.D. Wassenaar, Netherlands, Elsevier Press, Incorporated, 1959. $19.95.

The author, an experienced neurosurgeon from St. Ursula Clinic, has approached the problem of low-back pain from a fresh point of view. This book is well written, profusely illustrated with hand drawings, and divided into three parts.

Part I describes the changes in the spine that accompany degenerative disease and indicates how these may cause pain syndromes in the absence of demonstrable herniation of the nucleus pulposus.

Part II is devoted to a critical review of the world literature with careful correlation of pertinent data, both from English-speaking and non-English-speaking authors. It deals with the pre-

disposing factors of low-back pain—namely, the inherently weak back, congenital anomalies, joint changes, and variations in the vertebral canal and its content. The author discusses degeneration of the intervertebral disc as a part of the aging process.

Part III, larger than the other two sections combined, is devoted to the presentation and discussion of selected cases from over 2,000 explorations of the lumbar spine, selected to help differentiate low-back pain and radicular pain due to causes other than fresh prolapse of the intervertebral disc in young individuals. The author has placed special emphasis on the many possible causes of irritation of the portions of the spinal nerve roots that are between the dura and the dorsal root ganglion in the lateral recess of the intervertebral canal. Fresh prolapse of the intervertebral disc is only one of the many such causes. No attempt is made to calculate the frequency distribution of the etiological factors involved.

Although many of the ideas put forth by the author are largely theoretical and lack adequate documentation, his work serves a very great need in de-emphasizing the role of intervertebral-disc prolapse as the only cause of organic back complaints and attempts to place this condition in its proper perspective regarding complaints concerning the back. The careful organization and presentation of material is most thought-provoking and deserves careful consideration by all physicians, irrespective of their specialty, who treat many conditions of low-back pain with or without sciatica.

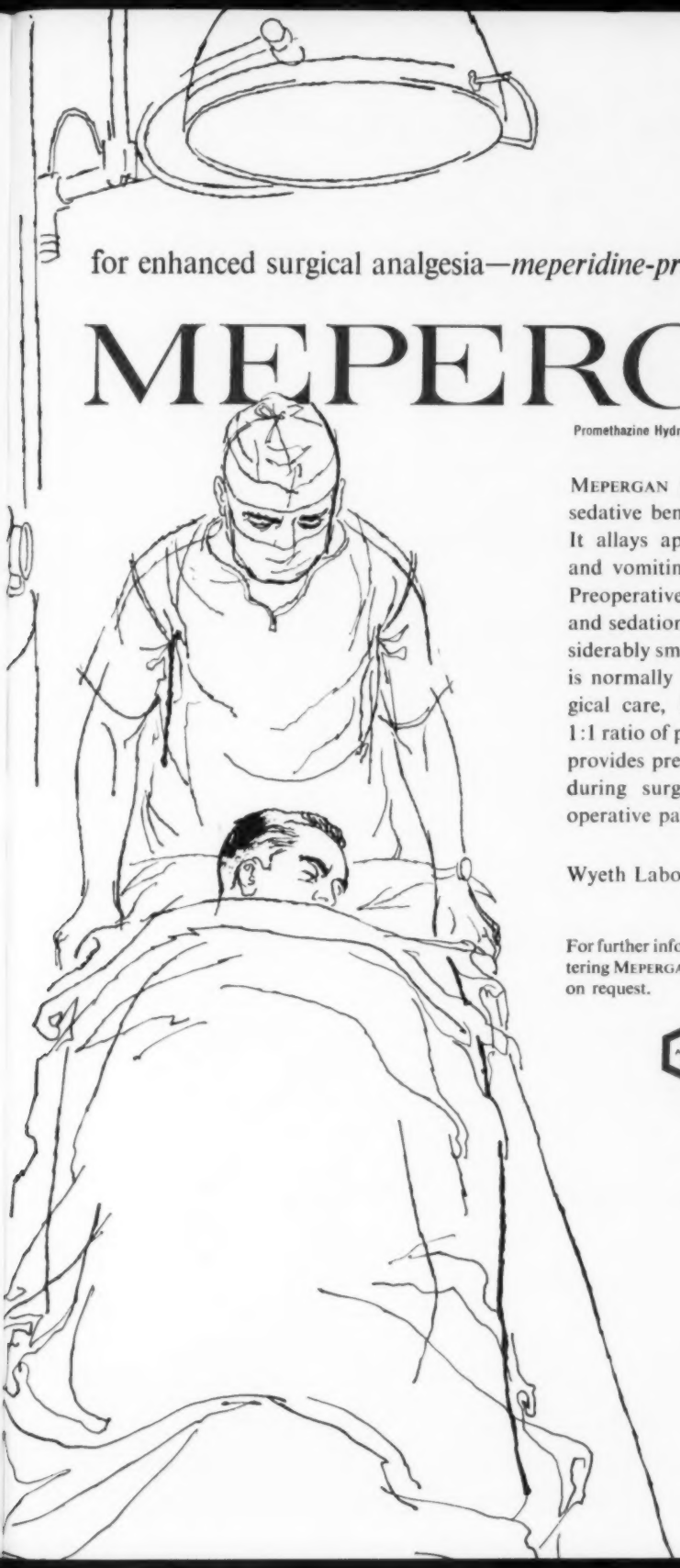
Harry C. Morgan, M.D.

ESSENTIALS OF ORTHOPAEDICS. Philip Wiles, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S. Third Edition. Boston, Little, Brown and Company, 1959. $13.00.

The work of the author of a textbook on surgery, like that of woman, is never done. This new edition of Mr. Wiles' book, first published in 1949, demonstrates, page after page, the continuous, and, one can hope, continuing process of revision, addition, and deletion; of illustrations added or replaced that keep such a book abreast of its subject. Sections extensively revised include postural defects of the feet, low-back pain, the anatomy and pathology of the intervertebral discs, spondylolisthesis, scoliosis, the surgery of osteo-arthritis of the hip, gout, diseases of the tendon sheaths, rheumatoid arthritis and tuberculous arthritis, and blood and allied diseases. New topics are congenital vertical talus, stress fractures, aneurysmal bone cysts, adamantinoma, leukemia, hereditary hemolytic anemia, sarcoidosis, and hypertrophic "osteoarthropy".

"Philip Wiles", wrote Dr. William A. Rogers, "possesses, to a remarkable degree, the capacity of recognizing the vital or essential elements of a surgical subject and of expressing them simply, concisely and lucidly."

J.A.R.



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SURGERY IN THE AGED

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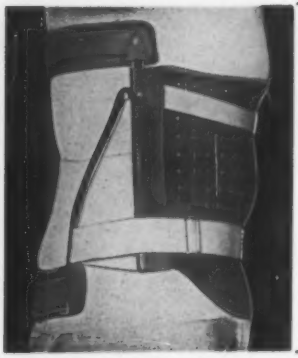

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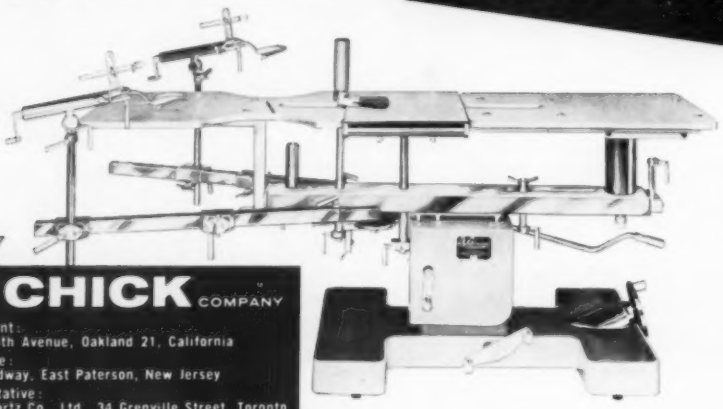

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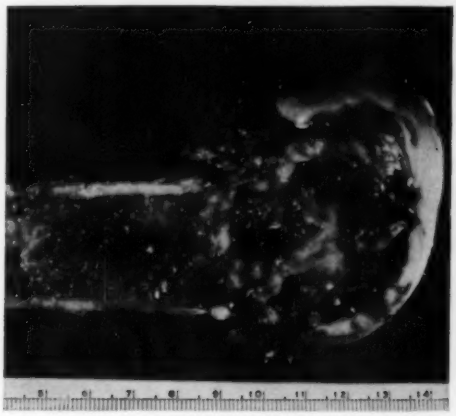

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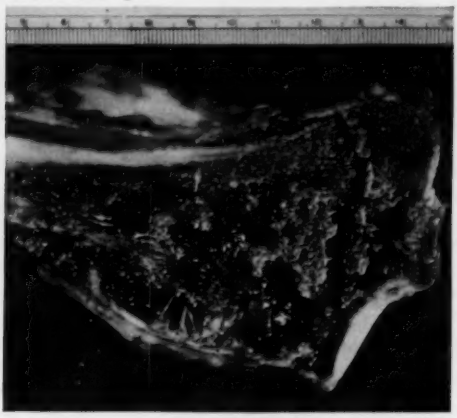

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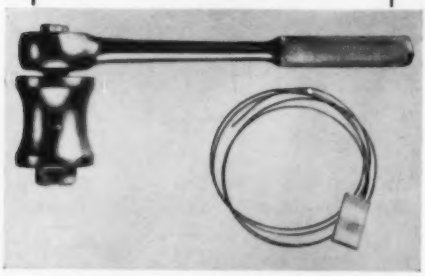

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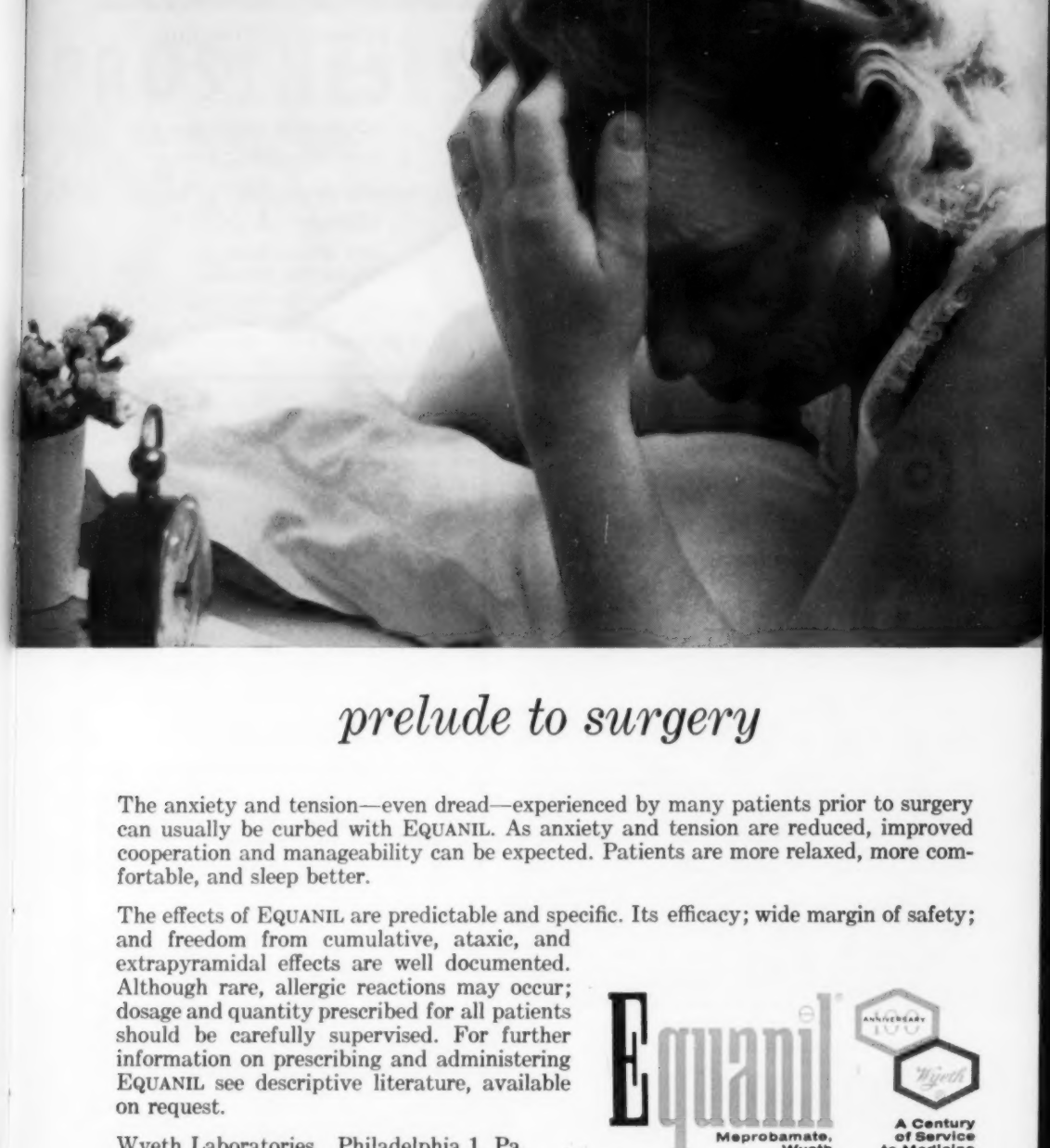

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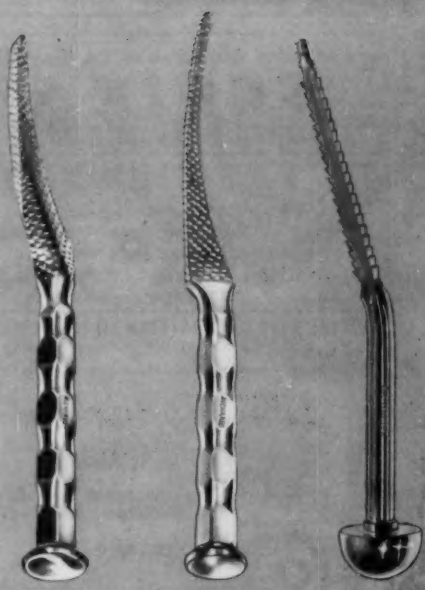

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ADDRESSES OF CONTRIBUTORS

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Yoshihiko Akahoshi, M.D., University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

C. E. Badgley, M.D., 1313 East Ann Street, Ann Arbor, Michigan

R. W. Bailey, M.D., 1313 East Ann Street, Ann Arbor, Michigan

Charles S. Ballinger, M.D., 206 West State Street, Rockford, Illinois

Joseph H. Boyes, M.D., 1401 South Hope Street, Los Angeles 15, California

Irvin Cahen, M.D., Louisiana State University School of Medicine, New Orleans 12, Louisiana

Jonathan Cohen, M.D., 300 Longwood Avenue, Boston 15, Massachusetts

Paul E. Dee, M.D., 1221 East State Street, Rockford, Illinois

William F. Donaldson, M.D., 121 University Place, Pittsburgh 13, Pennsylvania

Albert B. Ferguson, Jr., M.D., 125 DeSoto Street, Pittsburgh 13, Pennsylvania

Charles H. Fortson, M.D., 4100 W. Third Street, Dayton, Ohio

John J. Gartland, M.D., 269 South 19th Street, Philadelphia 3, Pennsylvania

Marvin C. Goodman, M.D., 29 Pearl Street, Lancaster, Pennsylvania

Miles J. Gullickson, M.D., 206 West State Street, Rockford, Illinois

Allan E. Inglis, M.D., 535 East 70th Street, New York 21, N. Y.

Joseph M. Janes, M.D., Mayo Clinic, Rochester, Minnesota

Patrick J. Kelly, M.D., Mayo Clinic, Rochester, Minnesota

Jerzy Kosowicz, M.D., 2nd Department of Internal Medicine of the Medical Academy in Poznań, Przybyszewskiego 49, Poznań, Poland

George E. Nelson, Jr., M.D., 416 Marquette Bank Building, Minneapolis 2, Minnesota

Lowell F. A. Peterson, M.D., Mayo Clinic, Rochester, Minnesota

Daniel C. Riordan, M.D., 1538 Louisiana Avenue, New Orleans 15, Louisiana

Ervin E. Rodriguez, M.D., 14 Valley Street, Lewistown, Pennsylvania

Russell N. Shroyer, M.D., 4100 W. Third Street, Dayton, Ohio

Michael Skovron, M.D., 716 Sassafras Street, Erie, Pennsylvania

James W. Smith, M.D., 232 East 66th Street, New York 21, N. Y.

R. Glenn Smith, M.D., 206 West State Street, Rockford, Illinois

C. B. Theodotou, M.D., Ohio State University College of Medicine, Columbus, Ohio

Claude E. Verdan, M.D., Av. de la Gare, 9, Lausanne, Switzerland

James N. Wilson, M.D., 1401 South Hope Street, Los Angeles 15, California

Jack L. Winters, M.D., Louisiana State University School of Medicine, New Orleans 12, Louisiana

Eduardo Zancolli, M.D., Santa Fé 954, 40–15, Buenos Aires, Argentina